Diseases of TREE FRUITS

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Alan L Jones

Turner Bond Sutton

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To The Grower

Year after year, disease-causing pathogens lower both yield and quality of apples, pears, peaches, cherries, and plums. This bulletin is designed to help the fruit grower achieve better control of these pathogens.

Proper recognition of the problem is the first step in successful and efficient control of tree-fruit diseases. Growers who can identify fruit diseases can select the most effective control measures. Whenever possible, photographs of symptoms, many in color, have been included as diagnostic aids. In addition, descriptions of spore stages of many pathogens are provided for microscopic identification.

Each disease has a developmental pattern or cycle it passes through during the year. Control chemicals and methods are generally effective only at certain times or stages in the disease cycle. If the pathogen is not in a susceptible stage of development when control is attempted, results will be disappointing. This bulletin outlines disease cycles as they are known to occur. Those portions of the disease cycle known to be important in developing successful control procedures are stressed.

Weather plays a critical part in determining the incidence and severity of diseases. Temperature, rainfall, and relative humidity can alter the development of the pathogen, the plant or both. Disease control programs must be adjusted annually according to prevailing environmental conditions to achieve the best results. Interrelationships between the pathogen, the host plant, and the environment are discussed in some detail as a guide in deciding when control procedures can be safely modified. For additional information, references are provided for many diseases. Most of the important diseases of tree fruits are described here. Specific chemical control recommendations for most of these diseases can be found in spray schedules published by the various states.

Pome Fruits

Apple Scab

Scab is caused by the fungus *Venturia inaequalis*, which occurs in most areas of the world where apples are grown. It is less severe in semi-arid regions than in cool, humid areas with frequent rainfall. The climate in all the fruit-growing regions east of the Rockies is extremely favorable for scab.

Symptoms

Apple scab occurs on the leaves, petioles, blossoms, and fruit. The most striking symptoms are on the leaves and fruit (Figs. 1, 2).

Infections usually develop first on the underside of leaves of fruit spurs, the side exposed when the fruit buds first open. Once the entire leaf has unfolded, both sides may be infected.

Severe infection can cause heavy defoliation. Trees defoliated 2 or 3 yr in a row are weakened and susceptible to low temperature damage.

Lesions develop as velvety-brown to olive spots which turn black with age. At first, the margins (edges) of the lesion are feathery and indefinite, but later distinct limits are evident.

Fruit infections resemble leaf infections when young but become brown and corky with age. Early scab infection results in uneven growth of the fruit and cracking of the skin and flesh. Lesions often develop around the blossom end of the fruit early in the season; later, they develop anywhere on the surface.

If infection occurs in late summer or early fall, rough black, circular lesions may develop on the fruit in storage. These lesions are usually small, varying in size from specks to ¼ inch diameter, and are known as "pin point" scab.

Pathogen Identification

Conidia are produced abundantly on new lesions on leaves (Fig. 3). They are one-celled, ovate to lanceolate, 12 to 22 μm by 6 to 9 μm. Ascospores are produced in flask-shaped fruiting structures termed perithecia (Fig. 4). As perithecia mature, they erupt through the leaf's cuticle, but remain partially buried in the leaf. If perithecia are removed, crushed on a microscope slide, and observed at 200X magnification, ascospores can be seen within club-shaped structures called asci. Mature ascospores are olive in color, two-celled, 11 to 15 μm by 5 to 6 μm.

Fig. 1. Scab on the leaves and fruit of McIntosh apple.

Fig. 2. Apple scab on the calyx end of McIntosh fruit, with small secondary lesions developing on the side.

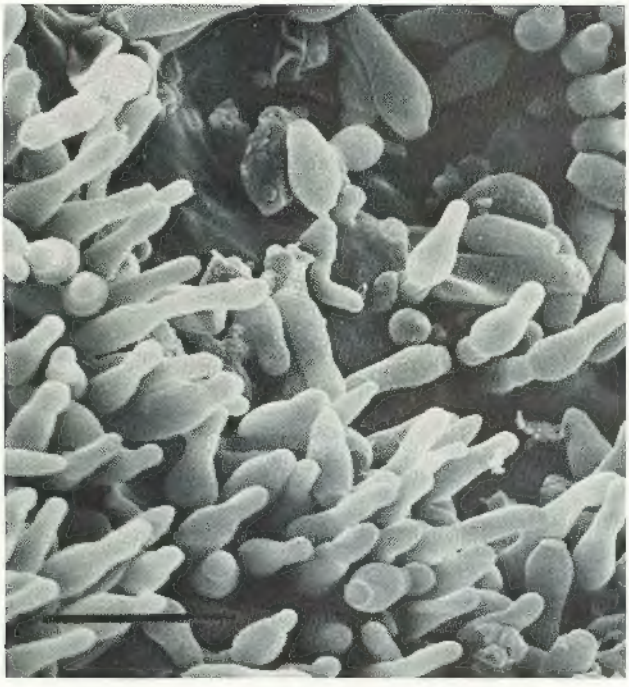

Fig. 3. Micrograph (microscopic enlargement) of a sporulating apple scab lesion on the surface of an apple leaf. Note pointed conidia being produced on short stalks or conidiophores. A few conidia are detached and lying on the leaf.

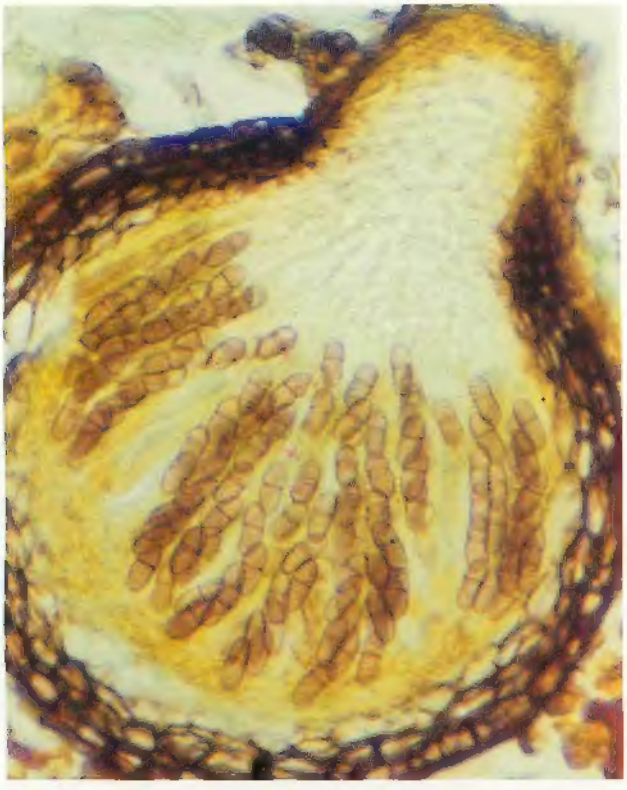

Fig. 4. Cross section of a perithecium of the apple scab fungus with pigmented mature ascospores in rows of eight ascospores per ascus *(courtesy J.R. James, CIBA-GEIGY, Greensboro, N.C.).*

Disease Cycle

Primary Cycle: The fungus overwinters in leaves on the orchard floor (follow Fig. 5 when reading the text on disease cycle). In late fall and early spring, microscopic, black, pimple-like structures, called perithecia, are produced in these dead leaves. Within each perithecium are asci, each with eight ascospores (Fig. 4). The ascospores produce the first, or primary, infections on the new growth.

Perithecial development is favored by alternating periods of wetness and dryness in late winter and early spring. Normally, some perithecia have mature ascospores when the blossom buds start to open. When the leaves on the orchard floor become wet, spores are forcibly ejected into the air. Air currents carry them to the emerging tissues where infection takes place. Maturation and discharge of ascospores usually lasts 5 to 9 wk.

Germination begins as soon as a spore lands on new, green leaves or fruit, provided a film of moisture is present (Fig. 6). The number of hours of wetting required for infection varies with prevailing temperatures. Growers should record the beginning

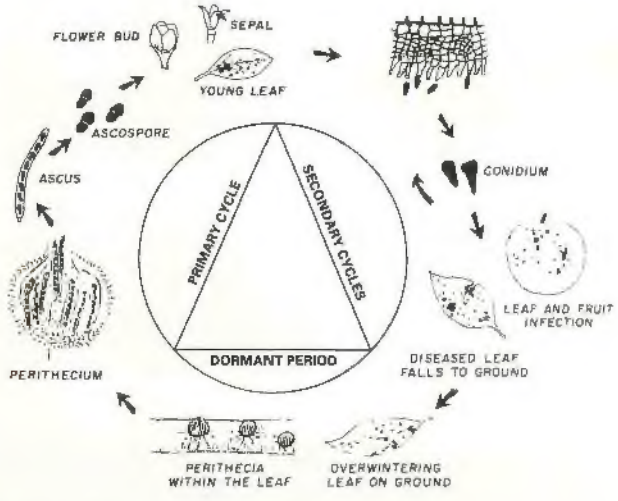

Fig. 5. Disease cycle of apple scab.

of rainfall and average temperatures, and determine the length of time it takes for infection to occur (Table 1). For example, at an average temperature of 58°F, primary infection will occur 10 hr after the start of the rain. After 22 hr of wetting, the degree of infection will be severe. This simple calculation is useful in deciding when fungicide sprays or dusts are needed.

Lesions are not visible until several days after the fungus first infects the leaf or fruit. The average

Table 1. Approximate number of hours of wetting required for primary apple scab infection at different air temperatures and the length of time required for development of conidia.*

Average Temperature	DEGREE OF INFECTION			Days Incubation[a]
	Light	Moderate	Heavy	
°F	hrs.[b]	hrs.	hrs.	days
78	13	17	26	
77	11	14	21	
76	9½	12	19	
63 to 75	9	12	18	9
62	9	12	19	10
61	9	13	20	10
60	9½	13	20	11
59	10	13	21	12
58	10	14	21	12
57	10	14	22	13
56	11	15	22	13
55	11	16	24	14
54	11½	16	24	14
53	12	17	25	15
52	12	18	26	15
51	13	18	27	16
50	14	19	29	16
49	14½	20	30	17
48	15	20	30	17
47	17	23	35	
46	19	25	38	
45	20	27	41	
44	22	30	45	
43	25	34	51	
42	30	40	60	
33 to 41[c]				

[a]*Approx. no days required for conidial development after primary scab infection.*

[b]*The infection period is considered to start at the beginning of the rain.*

[c]*Data are incomplete at low temperatures.*

**From W.D. Mills, Cornell University.*

temperature after penetration is important for determining the time required for visible lesion development. About 9 to 17 days are required from inoculation to the appearance of the olive-green, velvety scab lesions. Within the lesion are secondary spores (conidia) for perpetuating the disease in summer.

Secondary Cycle: Secondary infections are initiated by conidia produced in primary lesions. Since conidia may develop as soon as 7 to 9 days after infection, secondary infection, if not controlled, may be initiated if sufficient wetting occurs during bloom. This is particularly true when ascospores infect the apical portions of sepals and leaves at the silver tip stage of bud development. Conidia are disseminated

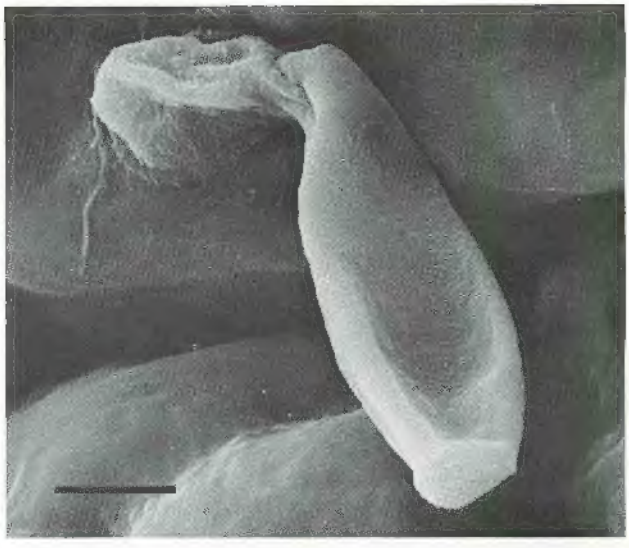

Fig. 6. Germination of a conidium of the apple scab fungus. The spore sends out a short germ tube with an appressorium at its tip from which the fungus penetrates the host tissue. Ascospores germinate similarly.

by splashing rain and by wind. Conidial germination and infection occurs under about the same conditions as for ascospores.

Secondary infection to fruit can occur in the fall but not show up until the fruit have been stored for several months. The disease can also build up on the leaves after harvest. Since the fungus overwinters in these leaves, perithecia may be present in quantity to start the new season even though a good spray program was followed the previous year.

Control

Resistant Varieties: Apple breeding programs to develop high-quality, disease-resistant varieties are being carried out at the New York State Experiment Station, Geneva and cooperatively between Purdue University, Rutgers University and the University of Illinois. Five scab-immune varieties, Prima, Priscilla, Sir Prize, Jonafree and Redfree, have been developed and released through the Purdue-Rutgers-Illinois program. Liberty and Freedom have been released through the New York program. Additional selections are in advanced stages of evaluation.

Sanitation: Prevention of perithecial formation in overwintering leaves would probably eliminate scab. Unfortunately, complete elimination of perithecia is not possible under orchard conditions with current methods. However, the potential for severe scab can be reduced by making applications of nitrogen to the foliage in autumn to hasten leaf decomposition.

Chemical Control: Apple scab is controlled primar-

ily with fungicide sprays or dusts. A variety of fungicides are available; how and when they should be used depends upon their mode of action.

Protectant fungicides prevent the spores from germinating or penetrating leaf tissue. However, to be effective, they must be present on the surface of susceptible tissues before infection. These chemicals may be applied periodically at pre-determined intervals, or according to anticipated rainfall (infection periods).

One protectant fungicide is effective in a single application technique. Rather than applying sprays every 5 to 10 days, a single spray is applied at the green tip stage of bud development. A high rate of fungicide is deposited on the branches, and through redistribution by rain, the new growth is protected for about 5 wk. After the fungicide deposit has weathered, a standard scab schedule is resumed.

Eradicant fungicides kill the fungus after infection has occurred. The relative ability of fungicides to eradicate scab is referred to as "back-action" or "kick-back" action. This is the number of hours that the chemical can be effectively applied after the infection period begins. Ratings of "kick-back" action are made for specific temperatures. The eradicant action for most fungicides is limited to a few hours or days after infection.

Today, most fungicides used for apple scab control are active as protectants and as eradicants. They control infections that may have occurred a few hours or days previous and also protect exposed tissue for several days after application. These compounds are usually applied at less frequent intervals, with timing depending on the weather and tree growth.

Systemic chemicals, which move into apple leaves, blossoms and green fruits, are also used for apple scab control. Because the fungicides are in the plant, they are less subject to weathering and therefore usually provide control for a longer period of time than protectant-eradicant compounds that are only on the leaf surface. Systemic compounds are frequently used to inhibit established lesions and to suppress spore production.

Resistance to Scab Fungicides: Resistance problems with apple scab have occurred with dodine (Cyprex) and benzimidazole fungicides (Benlate, Topsin M). Resistance develops where high and continuous selection pressure, such as from using one or closely related fungicides exclusively year after year, exists. Thus, benomyl-resistant scab occurs where benomyl is used regularly for about 3 yr, and dodine-resistant scab is likely where dodine is used for about 10 yr. Problems with fungicide resistance can be prevented by using non-related fungicides for part of the season or in a tank mixture with potentially troublesome materials.

HELPFUL REFERENCES

Jones, A.L. and J. D. Gilpatrick, eds. Apple and Pear Scab Workshop Proceedings. 1978. N.Y. State Agr. Exp. Sta. Special Rep. 28. 38 pp.

Sutton, T. B. and A. L. Jones. 1981. Apple disease management. Pages 257-273 in: Handbook of Pest Management in Agriculture. Vol. III. D. Pimentel, ed. CRC Press, Inc. Boca Raton, FL.

Fire Blight

Fire blight, caused by the bacterium *Erwinia amylovora*, is the major disease problem of pears. Blight is also a problem on susceptible apple varieties. The disease kills blossoms and fruiting spurs, and when severe, it kills branches, limbs and entire trees.

Symptoms

Infected blossoms become water-soaked and dark green as the bacteria invade the succulent tissues. Within a few days, the entire fruiting spur may be invaded (Figs. 7, 8). Infected tissues wilt and turn dark brown to black on pear; brown to dark brown on apple. Leaves may be infected through the petiole, resulting in discoloration of the mid-vein first, followed shortly by a darkening of the lateral veins and surrounding tissue.

Infected terminals wilt from the tip and often develop a crook or bend at the growing point. At first, the tissues are water-soaked and dark green, later turning brown to black. The disease sometimes progresses into the shoot from its base, blighting the lower tissues and girdling the parts above. Infected leaves and fruit often persist into winter.

Fig. 7. Fire blight blossom infection killing fruit spurs.

Fig. 8. Fire blight of apple. Note severe dieback of spurs and shoots.

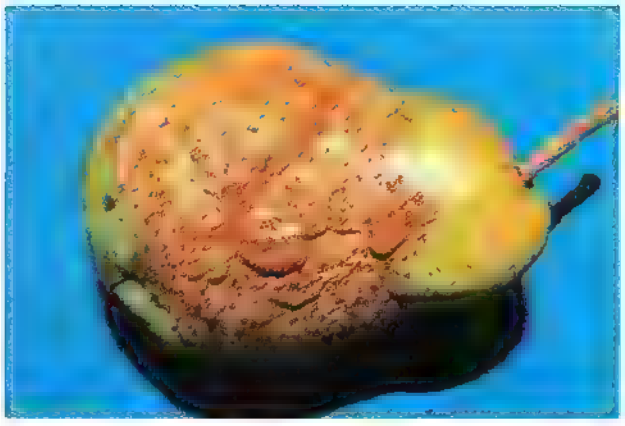

Fig. 9. Bartlett pear infected with blight. Note bacterial ooze.

Following infection, blight can move long distances within the living tissue and kill an entire tree in a season. Bark of invaded branches and scaffold limbs is darker than normal. When the outer bark is peeled away, the inner tissues are water-soaked with reddish streaks when first invaded; later the tissues are brown. When development slows down, the margins become sunken and sometimes cracked, forming a canker. The presence of reddening helps to distinguish fire blight cankers from low temperature damage to bark.

Apple and pear fruit infected with fire blight develop a brown to black decay (Fig. 9). The rotted areas remain firm, but the fruit eventually shrivel into mummies that persist on the tree.

During wet, humid weather, infected tissues often exude a milky, sticky liquid, or ooze, containing the fire blight bacteria. These droplets are produced in areas where the bacteria are actively multiplying. The ooze turns a reddish-brown upon exposure to air. Ooze on the surface of diseased blossoms, terminals, fruit or wood is the most obvious characteristic of blight.

Disease Cycle

Bacteria overwinter at the margins of cankers on branches. Survival is most likely in cankers with indefinite margins located on large branches. The proportion of these "holdover cankers" is highest following mild winters. Ooze may appear on the surface of the cankers beginning at the pre-bloom stage of bud development.

Before primary infection can occur, bacteria must move from overwintering cankers to the flowers. This is accomplished through the action of splashing rain and by flies and other insects which visit both the bacterial ooze and the blossoms.

Only a small portion of the blossoms are infected in this manner, provided care has been taken to eliminate overwintering cankers before growth begins. Eventually, however, honeybees visit diseased blossoms and pick up pollen or nectar contaminated with bacteria. Once this occurs, spread is rapid and infection severe. Secondary spread also occurs from splashing rain.

Climatic conditions are important in determining amount of spread and severity of blossom blight. Temperatures between 65 and 86°F favor infection. At the optimum temperature of 76°F, blossoms begin to show blight in 4 to 5 days. At lower temperatures, symptom development takes much longer.

Although bacteria invade the flower primarily through natural openings, wounds are important in the infection of terminal shoots, leaves and fruit. Water sprouts and shoots may be inoculated directly by piercing insects –primarily aphids and leafhoppers—or indirectly through the feeding wounds left by these insects. Wounds from hail stones are rapidly invaded by blight bacteria during the first 24 hr after injury occurs, leading to severe disease outbreaks. Under prolonged periods of wetness and high humidity, infection of leaves, shoots and fruit through natural openings may occur.

Control

Many practices help reduce the severity of fire blight. Not all measures are necessary or feasible in every planting. No single control method is adequate, however, and a conscious effort must be made to control the disease each year.

Removing sources of infection: In summer, periodically inspect orchards and remove infected spurs or terminals. This reduces spread of the disease and prevents extensive loss of bearing surface. Breaking

out infected twigs reduces the possibility of spreading the disease with the pruning tools. If infections are cut out, sterilize the tools with Clorox, diluted 1:10 with water. Remove at least 12 inches of normal-appearing bark below the lowest evidence of disease to insure elimination of the infection. Dormant pruning of all overwintering cankers is important. Make cuts about 4 inches below any evidence of dead bark killed by the disease.

Insect control: Sucking insects such as aphids, leafhoppers, plant bugs and pear psylla inoculate growing terminals and watersprouts and create wounds that bacteria can enter. Control these insects before bloom and throughout the summer.

Resistant varieties: Although varieties vary in resistance to fire blight, none are immune. Susceptible apple varieties include: Tompkins King, Twenty Ounce, Rhode Island Greening, Yellow Transparent, Jonathan, Ida Red, Rome, Puritan, Wealthy, Lodi, Fenton (Beacon) and many crab apple varieties. Some years, Golden Delicious, Delicious, McIntosh, and Stayman develop spur infections, but the disease seldom enters the main branches. M.26 and M.9 rootstocks are extremely susceptible to fire blight. Bartlett, Bosc and Clapp's Favorite pear varieties are highly susceptible.

When establishing new orchards, consider varietal susceptibility. Blight control is easier if plantings of susceptible trees can be isolated. Avoid interplanting susceptible apple varieties with pears or in fields adjacent to pear plantings. In mixed variety plantings, set varieties susceptible to blight in solid rows for ease of spraying with blight control chemicals.

Cultural practices: Lesion development and damage from blight is more severe when tree growth continues late into the season. Use management systems that promote early cessation of growth without excessively reducing tree vigor. Plant orchards on well-drained soil, apply nitrogen fertilizer early, and, to stop growth, cultivate no later than July 1.

Chemical control: Bactericides have been highly effective against the blossom phase of fire blight. These sprays are important because they often prevent fire blight from getting started. Once the disease is established, control is difficult. Although currently available chemicals have some effect on terminal blight, other chemicals and procedures are being studied to find more efficient methods to control this phase of the disease.

HELPFUL REFERENCES

Van der Zwet, T. and H. L. Keil. 1979. Fire blight. A bacterial disease of rosaceous plants. U.S. Dept. Agr., Handbook 510. 200 pp.

Rust Diseases

Rust diseases and causal fungi include: 1) cedar apple rust, *Gymnosporangium juniperi-virginianae;* 2) quince rust, *G. clavipes;* and 3) hawthorn rust, *G. globosum.* All three fungi spend part of their life cycle on red cedar and are problems only when red cedar is found close to the orchard. The life cycle and control of these diseases are similar. Cedar-apple rust is the most important

Symptoms

On Apple: The bright color of the lesions produced by cedar-apple rust makes it easy to identify (Fig. 10). Small, pale yellow spots develop on the upper leaf surface shortly after bloom. These spots gradually enlarge and turn orange. Orange-colored drops of liquid may be observed in the spots when they are about 1/8 inch in diameter. Later, black dots (pycnia) appear in the spot on the upper surface (Fig. 11). In late summer, cylindrical tubes or protuberances (aecia) become evident on the leaf undersurface (Fig. 12). The ends of the tubes split open and curl back.

Severe infection results in heavy defoliation and weakens the tree. Fruit infection is most common near the calyx end. Lesions are similar in color to those on leaves except the border is darker green. Lesions are shallow, not over 1/16 inch deep and with no internal chlorosis. The black pycnia often develop in the lesions, but formation of the cylindrical tubes is less common.

Hawthorn rust produces lesions on apple leaves only. Lesions resemble those of cedar apple rust but are usually not over 1/6 inch in diameter.

Quince rust does not cause leaf lesions. Lesions on fruit are dark green, about 3/4 to 1 1/2 inches in diameter, and usually distort the fruit (Fig. 13). Lesions are deep, with necrotic tissues extending to the

Fig. 10. Apple leaves and fruit with cedar-apple rust *(courtesy J.D. Moore, U. of Wisconsin).*

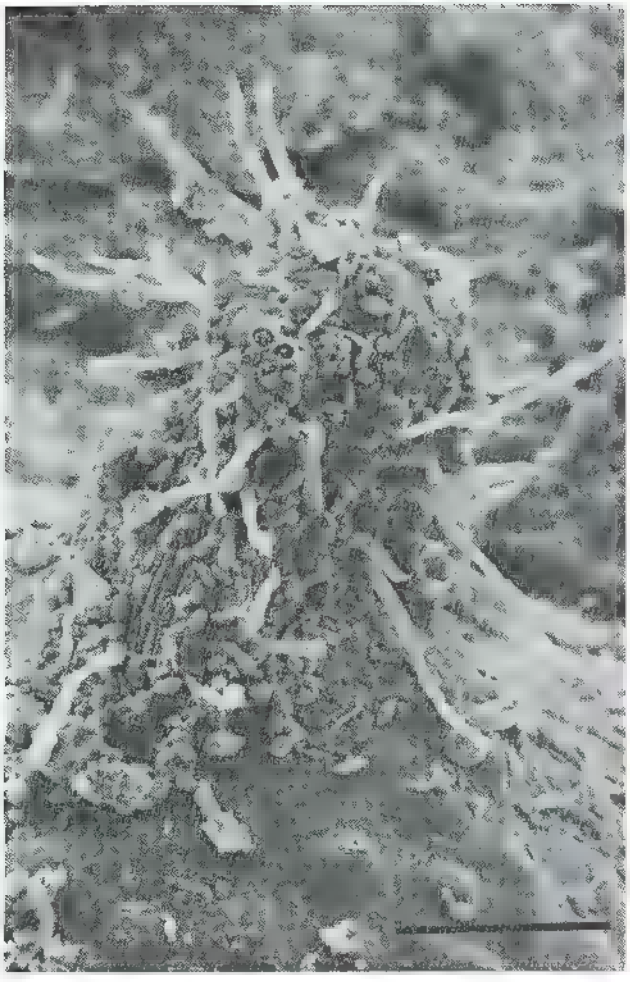

Fig. 11. Micrograph of a pycnium of cedar-apple rust fungus with a few periphyses extended and some pycniospores. Nectar drops were removed during preparation.

Fig. 13. Distortion of Delicious fruit infected with quince rust.

Fig. 14. Caylx end of Delicious fruit infected with quince rust and discoloration of flesh around the core.

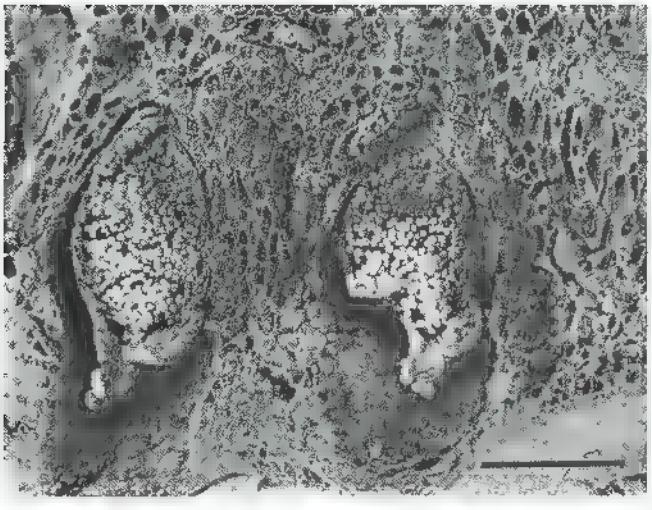

Fig. 12. Cross section of an apple leaf showing young aecia "cluster-cups" of the cedar-apple rust fungus (left) and a surface view of an aecium of hawthorn rust (right). Both are seen on the underside of the leaf.

core (Fig. 14). Affected fruit usually drop before harvest.

On Cedar: *Gymnosporangium juniperi-virginianae* and *G. globosum* produce brown to reddish brown leaf galls from 1/4 to 2 inches in diameter. During rainy periods in spring, bright orange, gelatinous, spore-horns protrude from the surface of the galls (Fig. 15). *Gymnosporangium juniperi-virginianae* galls produce spores for 1 yr; *G. globosum* may produce spores for 3 to 5 yr. *Gymnosporangium clavipes* produces spindle-shaped swellings (cankers) on branches of cedar (Fig. 15). These swellings may be up to 2 ft long and produce spores for many years.

Disease Cycle

The disease cycle of cedar-apple rust is complex (Fig. 16). Two plants (apple and cedar) and three fruiting structures (telia, aecia and pycnia) are involved. The pathogen requires 2 yr to complete its life cycle.

The fungus overwinters in reddish-brown galls or "cedar apples" in the cedar tree. When wet in spring, the galls extrude gelatinous tendrils or "horns" consisting of two-celled teliospores (Fig. 17). Each spore produces four basidiospores from each of the two cells. Air currents carry the basidiospores to the apple leaf and fruit where they infect within 4 hr under favorable conditions. Leaves are most susceptible when 4 to 8 days old. In July and August, windborne aeciospores from apple infect cedar leaves. Rust lesions develop in 1 to 3 wk. The second year after infection, these galls mature. In July and August, cedar-infecting aeciospores are produced which are carried back to cedar trees, thereby completing the life cycle of the fungus.

Control

Removal of cedars located within a 2-mile radius of the orchard interrupts the life cycle and makes fungicidal control easier. For complete control, remove all cedars within 4 to 5 miles of the orchard.

Several fungicides are highly effective against the rust diseases. They should be applied periodically from the pink stage of bud development to third cover to protect the emerging leaves and developing fruit.

Fig. 15. Cedar-apple rust gall on cedar with gelatinous spore-horns fully extended (top) and cankers of quince rust on cedar (bottom).

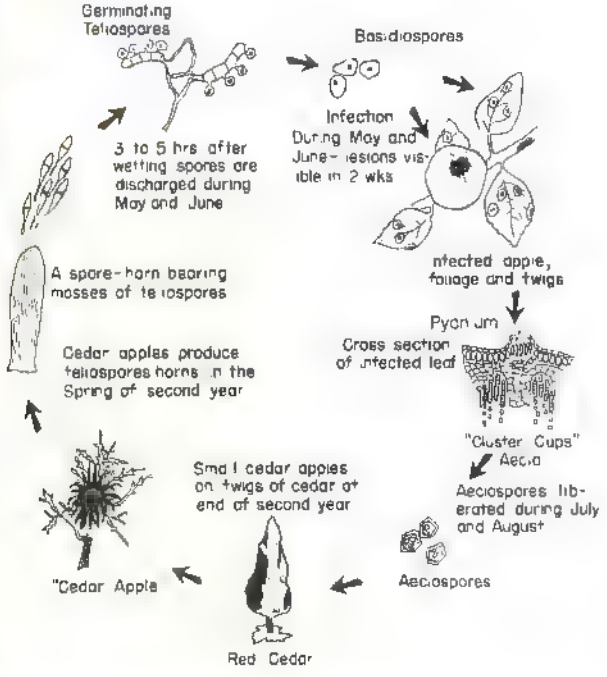
Germinating Teliospores
Basidiospores
3 to 5 hrs after wetting spores are discharged during May and June
Infection During May and June - lesions visible in 2 wks
Infected apple, foliage and twigs
A spore-horn bearing masses of teliospores
Pycnium
Cross section of infected leaf
Cedar apples produce teliospores horns in the Spring of second year
"Cluster Cups" Aecia
Aeciospores liberated during July and August
Small cedar apples on twigs of cedar at end of second year
"Cedar Apple
Aeciospores
Red Cedar

Fig. 16. Disease cycle of cedar-apple rust *(courtesy K.D. Hickey, Pennsylvania State Univ.).*

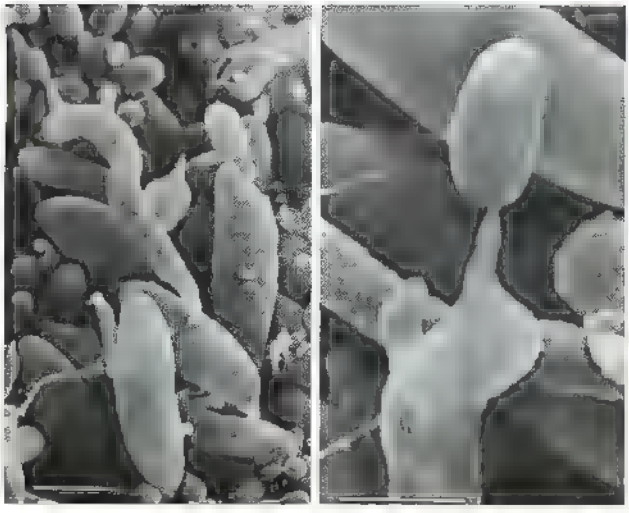

Fig. 17. Micrograph of a gelatinous spore-horn from a cedar-apple rust gall. Teliospores of the cedar-apple rust fungus with slight constriction at the crosswall and single teliospore bearing promycelium (left), and formation of secondary basidiospore on a pointed sterigmata (right). Other basidiospores can be seen on the left.

HELPFUL REFERENCES

Aldwinckle, H. S., R. C. Pearson, and R. C. Seem. 1980. Infection periods of *Gymnosporangium juniperi-virginianae* on apple. Phytopathology 70:1070-1073

Miller, P. R. 1932. Pathogenicity of three red cedar rusts that occur on apple. Phytopathology 22:723-740.

Miller, P. R. 1939. Pathogenicity, symptoms and causative fungi of three apple rusts compared. Phytopathology 29:801-811.

Powdery Mildew

Powdery mildew, caused by the fungus *Podosphaera leucotricha*, is often an important problem in apple orchards. Losses result from (1) death of vegetative shoots, (2) death of flower buds (and the resulting yield reductions), and (3) loss of fruit quality due to russeting. It usually is destructive only on highly susceptible varieties, but after it has built up to high levels, it may affect adjacent, moderately resistant varieties. The problem is greatest when a mildewcide is not included regularly in the scab program.

Symptoms

Mildew occurs in the nursery on terminal growth and in the orchard on leaves, flowers, shoots and fruit. On leaves, lesions first appear as whitish, felt-like patches of fungal mycelium and spores on the undersides and along the margins. The lesions spread rapidly, engulfing the entire leaf. Infected leaves are narrower than normal, folded longitudinally, and become stiff and brittle with age (Fig. 18).

Fig. 18. Powdery mildew infected shoot. Note distorted leaves and white growth on some leaves and along the shoot.

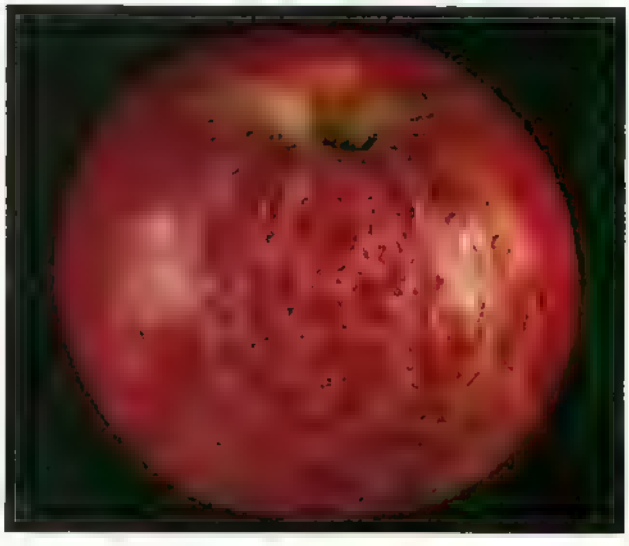

Fig. 19. Fruit showing net russeting from powdery mildew infection.

Infected blossom buds open several days later than normal or are killed outright by low winter temperatures. When they emerge, flower parts and leaves are usually covered with the white mycelium. Infected terminal or shoot buds also produce diseased leaves and shoots. Shoots from infected buds are shorter than those from healthy buds.

The fruit are rarely infected, except on Jonathan, unless the disease has built up to high levels. Infected fruit are russeted and sometimes dwarfed (Fig. 19).

Disease Cycle

The mildew fungus overwinters as mycelium in

buds infected the previous summer. As the infected buds open in spring, the fungus, already established on the emerging leaves, begins to produce spores (conidia). The conidia are produced in chains. They are carried by wind to the emerging tissues initiating secondary infections.

Unlike apple scab, which requires a film of moisture to germinate, mildew spores germinate readily in the absence of free moisture, at high relative humidities, and temperatures between 60 and 80°F. Spore germination and mycelial development are slow at temperatures between 40 and 50°F and slight at temperatures above 90°F. Mycelium from the germinating spores ramifies over the surface of the leaf, putting down small "roots", termed haustoria, onto the epidermal cells for nutrition. More spores are quickly produced, and the cycle is repeated. Secondary cycles continue to occur until tree growth stops in late summer. Small, dark brown, globular fruiting bodies (cleistothecia) are sometimes formed in the mycelial mat on stems and petioles in late summer (Fig. 20). Each cleistothecium has one ascus containing eight ascospores, but it is doubtful they play an important role in overwintering the fungus.

Mildew-infected buds are more susceptible to freezing than healthy buds. At −15°F or colder, most infected buds are killed. Because the mildew fungus is an obligate parasite, it can not survive after the buds are killed.

Fig. 20. Cleistothecia of *Podosphaera leucotricha* with long, unbranched appendages within the fungal mat of the mildew fungus on the surface of an apple leaf.

Control

Mildew-susceptible varieties include Jonathan, Rome Beauty, Cortland, Baldwin, Monroe and Ida Red. Where susceptible varieties are grown, include a mildewcide in the scab program to provide control of both diseases. Start sprays at tight cluster and continue until terminal growth stops. Early sprays (tight cluster to petal fall) are essential to success in controlling powdery mildew.

HELPFUL REFERENCES

Spotts, R. A. , R. P. Covey, and P. M. Chen. 1981. Effect of low temperature on survival of apple buds infected with the powdery mildew fungus. HortScience 16:781-783.

Sutton, T. B. and A. L. Jones. 1981. Apple disease management. Pages 257-273 in: Handbook of Pest Management in Agriculture. Vol. III, D. Pimentel, ed. CRC Press, Inc. Boca Raton, FL.

Black Rot

Black rot of apple fruit is caused by the fungus *Physalospora obtusa*. It is also called frog-eye leaf spot because of the appearance of the leaf lesion. In addition, the fungus causes a limb canker. The limb canker phase is most important in the northeastern and north central apple growing regions of the United States, and the leaf spot and fruit rot phase is most important in the southeast.

Symptoms

Leaf symptoms first appear 1 to 3 wk after petal fall. Leaf infections begin as small purple flecks which rapidly enlarge to 1/8 to 1/4 inch. The margins of the lesions remain purple, and the centers become tan to brown, giving the lesions a "frog-eye" appearance. Heavily infected leaves become chlorotic and abscise.

Sepal infection may occur prior to bloom, resulting in early fruit abortion or blossom-end-rot later in the season. After petal fall, infections on young fruit begin as reddish flecks which develop into purple pimples. These enlarge into dark brown necrotic areas as the fruit mature. Incipient infections on more mature fruit are often black, irregularly shaped, and surrounded by a red halo. As lesions enlarge they are often characterized by a series of concentric rings alternating from black to brown

Fig. 21. Golden Delicious apple with black rot developing on the calyx end.

Fig. 22. Black rot canker on an apple branch.

(Fig. 21). Lesions remain firm and are not sunken. Pycnidia are usually scattered over the surface of the lesion. Infected fruit mummify and often remain attached to the tree.

Infected areas on limbs and branches are reddish brown and slightly sunken (Fig. 22). Some cankers remain small, others enlarge to become several feet in length. Infected branches are sometimes weakened enough to break with heavy crop loads; sometimes they are killed outright.

Pathogen Identification

Pycnidia are produced abundantly on limb cankers, in dead twigs, occur on fruit, and are occasionally produced in leaf spots. Conidia are brown, usually nonseptate, ellipsoidal to pyriform and average 12 by 25 μm. Perithecia are often found in dead wood and cankers. Ascospores are ellipsoid 11 to 15 by 23 to 34 μm and appear to have thickened walls.

Disease Cycle

Physalospora obtusa overwinters in dead bark, twigs, cankers and mummified fruit. Ascospores and conidia are released during rainfall throughout the growing season and are washed or blown onto fruit and foliage. Ascospores are generally more common during the spring than summer months. Sepal infection can occur any time after bud break, and fruit infection can occur any time during the growing season. Leaf infection is most common just after petal fall. Black rot infection of leaves and fruit commonly develops in cone-shaped areas beneath black rot mummies or old fire blight cankers. Early-season infection may result in fruit drop. Severely diseased fruit may mummify and remain attached to the tree.

Control

Removal of dead wood, mummies and cankers is important to help control the disease. Remove prunings piled on the orchard's perimeter, as these are important inoculum sources. Fungicides, applied from silver tip until harvest are required to control the disease when it is a problem.

HELPFUL REFERENCES

Foster, H. H. 1937. Studies of the pathogenicity of *Physalospora obtusa*. Phytopathology 27:802-827.

Holmes, J. and A. E. Rich. 1969. The control of black rot cankers on apple trees in New Hampshire. Plant Dis. Rep. 53:315-318.

Taylor, J. 1955. Apple black rot in Georgia and its control. Phytopathology 45:392-398.

White Rot

White rot, caused by the fungus *Botryosphaeria dothidea*, is also referred to as Bot Rot or Botryosphaeria rot. The disease is most common in the southeastern apple-growing regions of the United States, where losses in some orchards have exceeded 50%.

Symptoms

Fruit lesions begin as small, often circular, slightly sunken, tan spots, which may be surrounded by a red halo (Fig. 23). On varieties with red skin pigment, the halo may appear purple or black. As lesions expand, the rotten area extends in a cylindrical manner toward the core (Fig. 24). In more advanced stages, the core becomes rotten, and the rotten area advances from the core region until the entire fruit is rotten. Under warm conditions (80°F or more) rotten areas are usually soft, watery and clear to tan in color. If rotten areas develop under cooler conditions they are usually firmer, a deeper tan and resemble black rot. Scattered clumps of pycnidia develop on the surface of affected fruit. Rotten fruit usually drop, but a few may shrivel and remain attached to the tree.

Branch and twig infections begin as a discoloration of the lenticels that later develop into blisters, which may exude a liquid (Fig. 25). In older cankers, the outer bark becomes tan to orange and papery, and the margins of the cankers crack and fissure. Large limbs may be killed by fusion of cankers. Pycnidia and perithecia are produced in the canker area.

Pathogen Identification

The *Dothiorella*, or asexual stage of *B. dothidea*, is the most common stage on infected fruit, cankers or dead wood. Conidia produced in pycnidia are hyaline, one-celled, fusoid and 12 to 30 by 4 to 8 μm. Perithecia are often produced in twig cankers or in dead wood. Ascospores are one-celled, elliptic, hyaline and 15 to 24 by 6 to 10 μm. A gelatinous coating is sometimes attached to the ascospores.

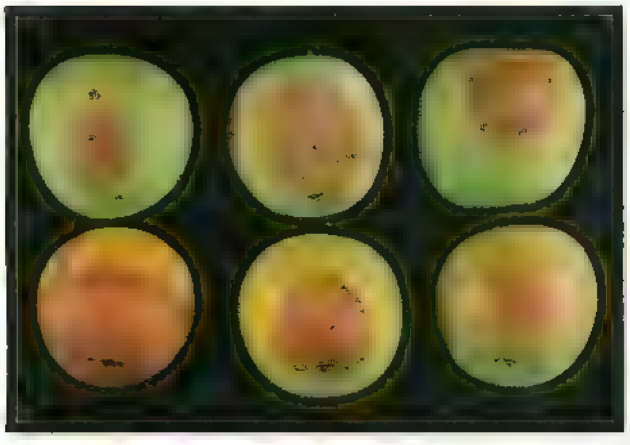

Fig. 23. Several examples of white rot on Golden Delicious fruit. Apple lower left infected with black rot.

Disease Cycle

Botryosphaeria dothidea overwinters in dead bark, twigs and cankers within the tree. Ascospores and conidia are produced throughout the growing season and are washed or blown onto fruit or twigs during rainy periods. Fire-blighted branches and spurs and other dead wood are rapidly colonized and can be an important source of inoculum during the growing season. Although pycnidia are produced on infected fruit, spores produced in them contribute little to disease incidence during the growing season.

Fruit infection can occur throughout the growing season, but rot symptoms usually do not appear before fruit begin to mature. Infection occurs primarily through wounds. Infection of branches

Fig. 24. Internal fruit symptoms of apple white rot (right) and bitter rot (left). Note that decay from white rot reaches the core or center of the fruit.

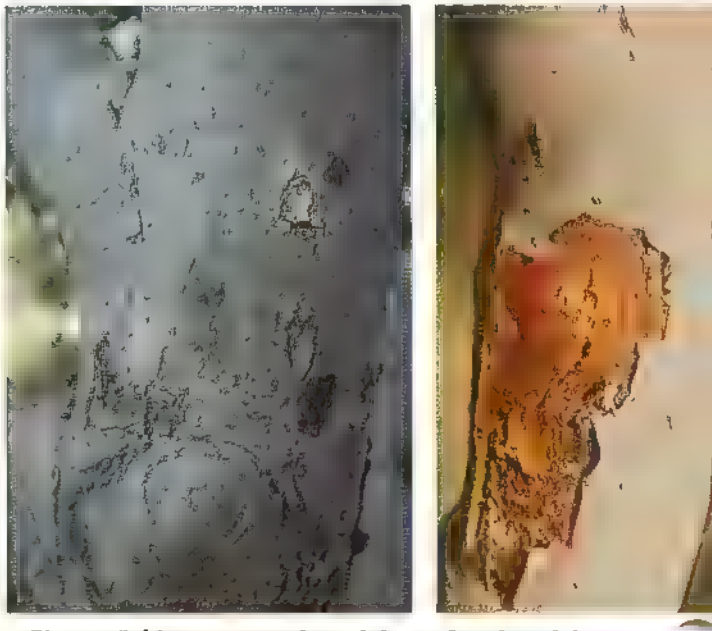

Fig. 25. White rot. Branch on left exuding liquid from blisters. In photo on right, the papery outerbark has been removed to show rings with fungus fruiting bodies. *(courtesy P.A. Arneson, Cornell Univ.)*

occurs through wounds or lenticels. Bark injured by temperature stress is an important infection site. Weak trees exhibiting poor growth are often more susceptible to bark infection.

Control

Because *Botryosphaeria* lives and develops in dead wood, pruning and removing all dead spurs, twigs and branches from the orchard is essential. Removal of current season fire-blighted twigs is also important since they can be rapidly colonized by the white rot fungus.

A fungicide spray program from bloom until harvest is important to assure white rot control. The ethylene-bis-dithiocarbamate fungicides are not particularly effective. Trunk cankers can be controlled with spring and fall fungicidal sprays.

HELPFUL REFERENCES

Connor, S. R. 1968. Canker formation on apple bark by *Botryosphaeria ribis*. Ph.D. Thesis, University of Delaware, Newark. 157 pp.

Fulkerson, J. F. 1960. *Botryosphaeria ribis* and its relation to a rot of apples. Phytopathology 50:394-398.

Kohn, F. C., Jr. and F. F. Hendrix. 1982. Temperature, free moisture and inoculum concentration effects on the incidence and development of white rot of apple. Phytopathology. 72:313-316.

Bitter Rot

Bitter rot is the common name of the fruit rot disease caused by *Glomerella cingulata*. The fungus also causes a leaf spot and canker. The disease is most common in apple-growing regions with temperatures warm enough to favor disease development. Of the three rot diseases, bitter rot, white rot and black rot, bitter rot has the potential to be the most destructive.

Symptoms

Fruit rot symptoms differ somewhat, depending on whether infection is initiated by spores from perithecial strains (strains which produce ascospores and conidia) or conidial strains (strains which produce only conidia). Initial symptoms produced by perithecial or conidial strains are similar.

Lesions begin as small, slightly sunken areas, which are light brown to dark brown. On mature fruit, lesions may be surrounded by a red halo. Lesions originating from infections by conidial strains remain circular and become sunken as they enlarge.

Copious quantities of conidia are produced in acervuli, which occur in concentric circles around the point of infection (Fig. 26). Acervuli are sparse on some lesions and very dense on others. Under moist, humid conditions, the spore masses appear creamy and are salmon to pink.

Fig. 26. Golden Delicious apple with several bitter rot lesions. Note the sunken nature and presence of spores in some of the lesions.

Fig. 27. Golden Delicious apple infected with a perithecial strain of the bitter rot fungus. Lesions are usually not sunken and are often darker brown than those caused by conidia.

Lesions initiated by perithecial strains are usually not sunken and are often darker brown than those caused by conidia. Acervuli are widely scattered over the surface, and perithecia are found in dark brown to black clumps scattered on the surface (Fig. 27).

Lesions initiated by perithecial and conidial strains extend in a cone shape manner toward the core. In cross section, the lesion appears "V" shaped

Fig. 28. Cross section of bitter rot infected fruit. Note "V"-shaped lesion.

(Fig. 28). This reasonably reliable characteristic can be used to separate bitter rot from white rot or black rot. The rotten area is brown but much firmer than white rot. Infected fruit mummify, and some may remain attached to the tree through the winter.

Leaf lesions are caused by the perithecial strain. They begin as small, red flecks, which enlarge to irregular brown spots 1/16 to 1/2 inch in diameter. Severely affected leaves may abscise.

Bitter rot cankers are rare in the eastern United States. Cankers are oval, sunken and often zonate in appearance.

Pathogen Identification

Conidial strains are easy to identify because of the copious conidia produced in acervuli on the surface of lesions (Figs. 29, 30). In mass, they are pink to salmon in color. Under the microscope, conidia are one-celled hyaline and vary in shape and size averaging 12 to 16 by 4 to 6 μm (Fig. 31). Clumps of perithecia are often produced on fruit infected with the perithecial strain. Ascospores are hyaline, slightly curved, unicellular, and 12 to 22 by 3.5 to 5 μm.

Disease Cycle

Glomerella cingulata primarily overwinters in dead wood or mummies in the tree. Other inoculum sources include stems of fruit which were torn from them at harvest or fruit mummied by chemical thinners. Conidia, produced in these overwintering sites, are the primary inoculum source in the spring, although ascospore inoculum is important in some orchards. Conidia are spread by rain; insects and birds are also involved in their dispersal. Ascospores are released by rain and are airborne. Fruit are susceptible from 3 wk after petal fall until harvest. Temperatures of 80 to 90°F are most favorable for disease development. Because of the large number of conidia produced in lesions on fruit, *G. cingulata* has the potential for rapid spread within the orchard.

Control

Removal of mummied fruit, dead wood and fire-blighted twigs is important to help control the disease. Fungicides, applied on a 10- to 14-day schedule from petal fall until harvest, are the most important means of control. More frequent applications may be necessary under conditions favorable for disease development. Removal of newly infected fruit from trees during the growing season will aid in control.

HELPFUL REFERENCES

Roberts, J. W. and L. Pierce. 1935. Apple bitter rot and its control. U.S. Dept. Agr. Farmers Bull. No. 938, 10 pp.

Shane, W. W. and T. B. Sutton. 1981. The *Gomerella cingulata* perfect stage and apple bitter rot. Plant Dis. 65 135-137

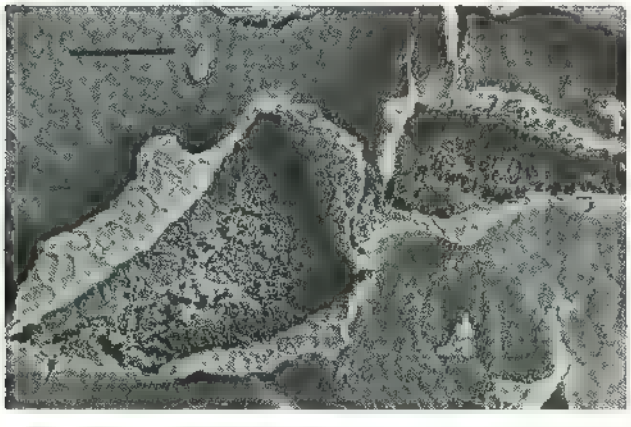

Fig. 29. Acervuli of *Colletotrichum gloeosporioides*, the imperfect stage of the bitter rot fungus, erupting through the epidermis of apple fruit.

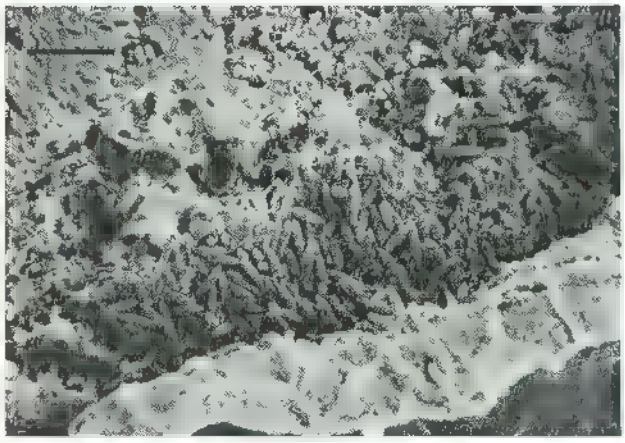

Fig. 30. Conidia of *Colletotrichum gloeosporioides* within an acervulus.

Sooty Blotch and Fly Speck

Sooty blotch and fly speck are common names of two diseases often found on apple and pear fruit at the same time. They are caused by different fungi: sooty blotch by *Gloeodes pomigena* and fly speck by *Microthyriella rubi*. They do little or no actual damage to the fruit, but their presence on the fruit's surface lowers quality and the subsequent market value. The diseases occur throughout the eastern apple-growing areas of the United States, but are most severe in the southern fruit-growing regions. Both fungi also occur on the bark, stems and leaves of many plant species with waxy cuticles.

Symptoms

Sooty blotch appears as superficial sooty or cloudy blotches on the surface of fruit. Lesions are brown to olive green and indefinite in outline (Figs. 32, 33). Four mycelial types have been identified on fruit. Lesions produced by them range from sooty smudge-like colonies to much darker colonies with many small, circular mycelial clumps, known as plectenchymal bodies, scattered within them.

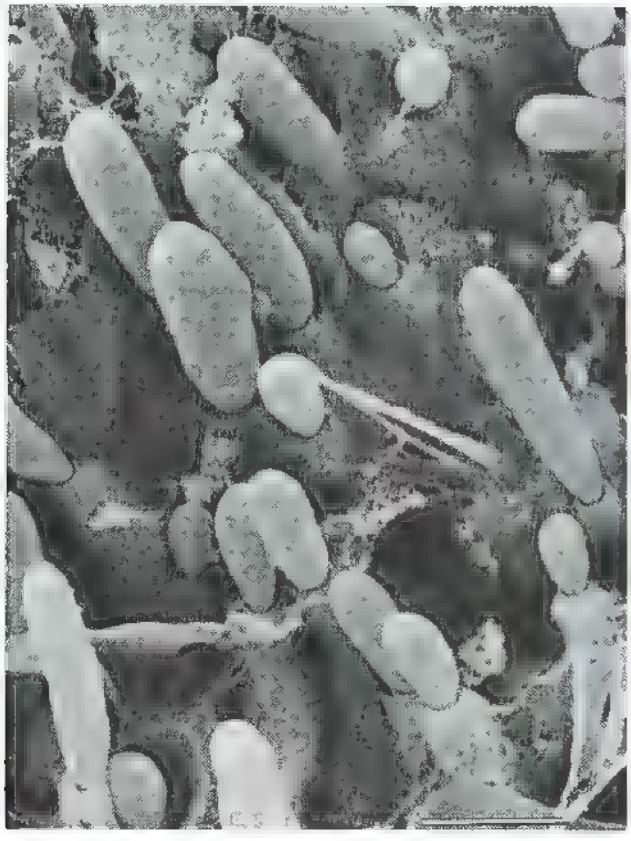

Fig. 31. Conidia of *Colletotrichum gloeosporioides* extruding from phialides within the acervulus.

Fly speck appears on fruit as sharply defined black shiny dots in groups of a few to 100 or more (Figs 32, 33). These dots or specks are sexual fruiting structures, termed ascocarps. They are much larger than the plectenchymal bodies associated with some sooty blotch mycelial types.

Pathogen Identification

These two diseases are recognized primarily by the macroscopic symptoms on the surface of infected fruit. The two pathogens are somewhat difficult to culture and often do not sporulate readily in culture. Ascospores of *M. rubi* can be found in the ascocarps in the late spring and early summer. Ascospores are 2-celled, hyaline, and range in size from 10 to 14 by 3 to 5 μm.

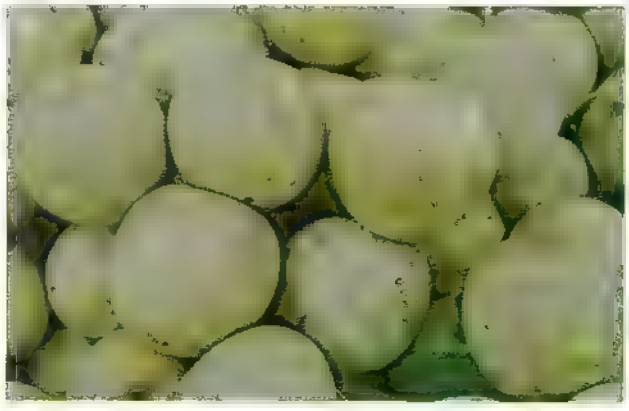

Fig. 32. Sooty blotch and fly speck on the surface of Golden Delicious apple *(courtesy K.D. Hickey, Pennsylvania State Univ.).*

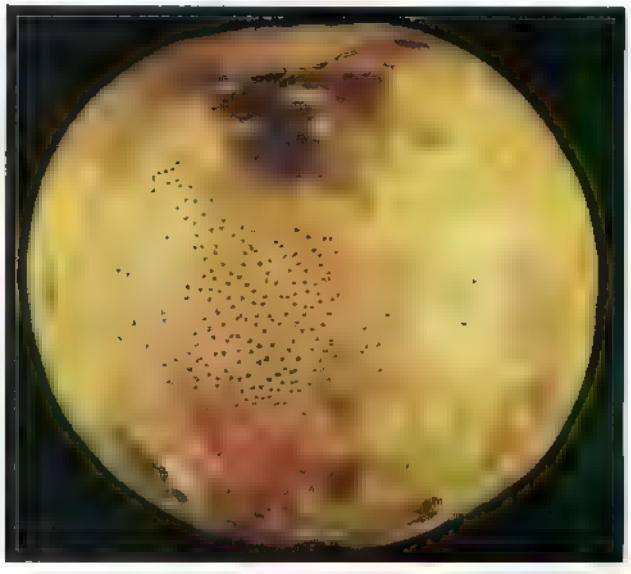

Fig. 33. Fly speck and sooty blotch on the surface of Rome Beauty apple.

Disease Cycle

Both pathogens overwinter on twigs of many woody plants. *Gloeodes pomigena* is spread from overwintering hosts by waterborne conidia or mycelial fragments. Spread of *M. rubi* is by airborne ascospores, which are discharged during rain periods or by airborne or waterborne conidia. Fruit infection can occur anytime after petal fall but is most prevalent in mid- to late-summer. Both diseases are favored by moderate temperatures, high humidity and abundant rainfall. *Gloeodes pomigena* and *M. rubi* are primarily restricted to the fruit's cuticle. Fruit severely affected with sooty blotch may shrivel more readily in storage.

Control

Control is achieved through sanitation and the use of fungicides. Removal of alternate hosts on the orchard's perimeter will help reduce the inoculum level. Good pruning to facilitate drying is very important in controlling the diseases. Fungicides applied on a regular schedule for "summer disease" control will usually control both diseases. The diseases are very difficult to control in areas in the orchard which are slow drying because of dew or fog. In these areas, fungicides may have to be applied more frequently than every 14 days.

HELPFUL REFERENCES

Baines, R. C. and M. W. Gardner. 1932. Pathogenicity and cultural characters of the sooty blotch fungus. Phytopathology 22:937-952.

Baker, K. F., L. H. Davis, R. D. Durbin, and W. C. Snyder. 1977. Greasy blotch of carnation and flyspeck of apple: Diseases caused by *Zygophiala jamaicensis*. Phytopathology 67:580-588.

Grove, A. B. 1933. A study of the sooty blotch disease of apple and the causal fungus *Gloeodes pomigena*. Va. Agr. Exp. Sta. Bull. 50. 43 pp.

Pear Scab

Pear scab occurs sporadically in isolated Bartlett pear orchards but frequently in backyard plantings with little known or exotic pear varieties.

Pear scab symptoms are similar to those of apple scab (Fig. 34). Unlike apple scab, however, twig infections are common and can be damaging. Twig infections and perithecia produced in the fallen leaves are the source of primary inoculum in spring. There are several strains of the pear scab fungus, *Venturia pirina*. Because strains of the fungus differ in the varieties they infect, resistant varieties in one region may not be resistant in another.

Fig. 34. Pear scab on fruit and leaves.

Control

Many of the fungicides that control apple scab also control pear scab. Check label clearance, however, before using a particular compound.

Fabraea Leaf Spot

Fabraea leaf spot (*Fabraea maculata*) is a fungus disease that affects both pears and quinces. Where the disease is severe, substantial defoliation may occur by mid-summer, thus weakening the tree. Severely infected fruit are often cracked, misshapen and unmarketable.

Symptoms and Disease Cycle

Fabraea spots can occur on leaves, shoots and fruit. The spots first appear as small, purplish-black dots and gradually enlarge to form circular, brown lesions about 1/8 to 1/4 inch in diameter (Figs 35, 36). A small, black pimple, or acervulus, develops in the center of each lesion from which the spores ooze in a creamy white mass in wet weather (Fig. 37). The spores (conidia) are very distinctive. They are composed of four cells; two small lateral cells are present on either side of the juncture of two larger ones. Setae formed at the apex of each cell give the spores an insect-like appearance (Fig. 38). The imperfect stage is *Entomosporium maculatum*.

The fungus overwinters both on twig cankers and on dead leaves on the ground. The spores are disseminated by washing and wind-blown rain.

Control

Fabraea spot is controlled by applying protectant fungicides beginning before bloom and continuing on a regular schedule until harvest.

Fig. 35. Fabraea leaf spot infection to pear leaves.

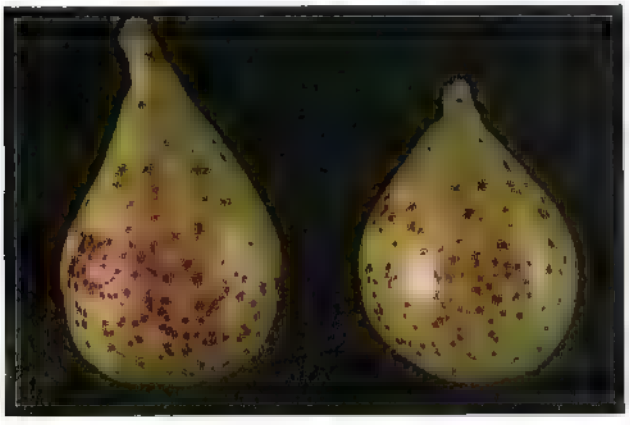

Fig. 36. Fabraea leaf spot infection to fruit of Tennessee Selection #345298.

Blister Spot

Blister spot of apple is caused by the bacterium *Pseudomonas syringae* pv. *papulans*. The disease is known to occur in several north central states, in New York state, and in Ontario, Canada. It is an important problem on the cultivar Mutsu. Although the disease has been reported on numerous other varieties, it is seldom of economic importance on these varieties.

Symptoms

Blister spot symptoms (Fig. 39) first appear in mid- to late-June in southern fruit growing areas and mid- to late-July in northern areas. Lesions are raised, brown to black blisters on the surface of the fruit. Young lesions may appear water-soaked. Older lesions appear to erupt as the fruit expand and mature. Lesions are 1/16 to 1/4 inch in diameter, 1/8 to 9/16

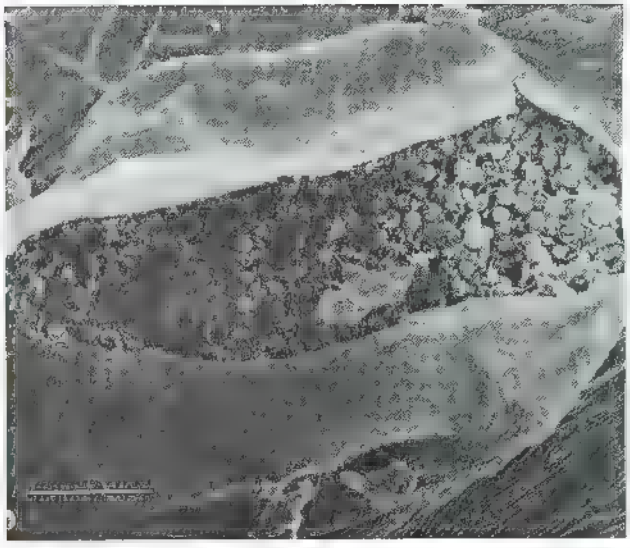

Fig. 37. Surface view of an acervulus of *Entomosporium maculatum* breaking through the epidermis.

Fig. 38. A conidium of Fabraea leaf spot with two lateral cells on either side of the juncture of two larger ones. Note setae or hair-like structures attached to the cells.

Fig. 39. Blister spot on maturing Mutsu apple.

inch deep. A red to purple zone of discoloration often develops in the skin bordering older lesions.

Pathogen Identification

Masses of rod-shaped motile bacteria can be observed microscopically in sections of infected tissue, particularly those of young lesions. Although the bacterium can be isolated on a number of common bacterial culture media, a selective differential medium (PSM) is superior to the common media because *P. syringae* pv. *papulans* can be distinguished from *P. syringae* pv. *syringae* based on colony morphology. Physiological and biochemical tests useful in confirming the identity of *P. syringae* pv. *papulans* are available.

Disease Cycle

The bacteria overwinter in buds and possibly in infected fruit on the orchard floor. In spring and summer, the bacteria colonize the leaves, blossoms and fruit without causing symptoms. Starting 2 to 2 1/2 wk after petal fall and continuing for 2 to 4 wk, the fruit are highly susceptible to infection through natural openings called stomata. As the fruit expand, the stomata are lost and infection declines. Results of artificial inoculations indicate lesions should appear about 10 to 12 days after initial infection.

Natural infection of Mutsu may involve a few to nearly all the fruit in the orchard. Although natural infection is rarely a problem on other varieties, the disease has been produced by artificial inoculation, and natural infections have been observed on Jonathan, Delicious, Golden Delicious and Rome Beauty. Attempts to produce the disease with *P. syringae* pv. *syringae* have been unsuccessful.

Control

Blister spot on the cultivar Mutsu can be controlled with well-timed antibiotic sprays during the period the fruit are susceptible. The first spray should be applied no later than 2 wk after petal fall and should be followed weekly by two additional sprays.

HELPFUL REFERENCES

Burr, T. J. and B. Hurwitz. 1981. Seasonal susceptibility of Mutsu apples to *Pseudomonas syringae* pv. *papulans* Plant Dis. 65:334-336

Burr, T. J. and B. Katz. 1982. Evaluation of a selective medium for detecting *Pseudomonas syringae* pv. *papulans* and *P. syringae* pv. *syringae* in apple orchards. Phytopathology 72:564-567.

Blossom End Rot

Blossom end rot is a sporadic problem that may infect 5 to 10% of the fruit in an orchard. The disease is usually caused by *Sclerotinia sclerotiorum*, but similar symptoms are caused by *Botrytis cinerea* and some other fungi.

Symptoms and Disease Cycle

Affected fruit become visible in the orchard about 1 mo after petal fall. A small, 1/4 to 1/2 inch area of brown rotted tissue develops adjacent to the blossom end of the fruit (Fig. 40). If the disease is caused by *S. sclerotiorum*, the rot is soft and often expands until one-third or more of the fruit is involved (Fig. 41). The rot associated with *Botrytis* infection is dry and shallow, and the rotted areas usually cork over well before harvest. The rotted area is often surrounded by a red border.

Presumably, *S. sclerotiorum* overwinters as sclerotia associated with dandelion, wild clover and possibly other plants in the orchard sod. Apothecia (Fig. 42) are produced in early spring and release asco-

Fig. 40. Botrytis blossom end rot of McIntosh apple.

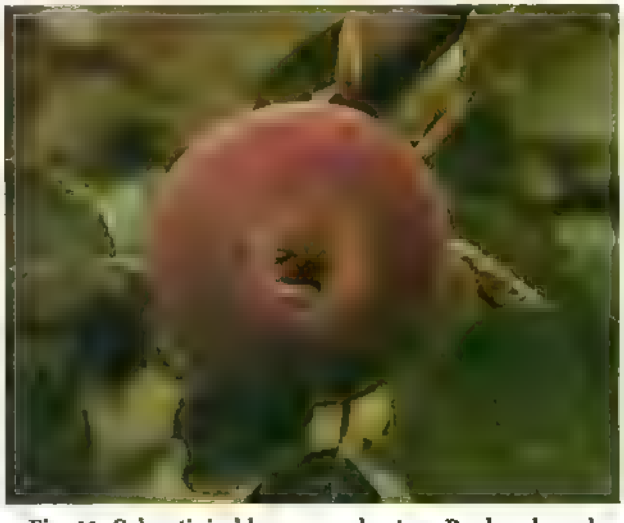

Fig. 41. Sclerotinia blossom end rot on Paulared apple.

Fig. 42. Apothecia of *Sclerotinia sclerotiorum (from MSU Extension Bulletin E-892).*

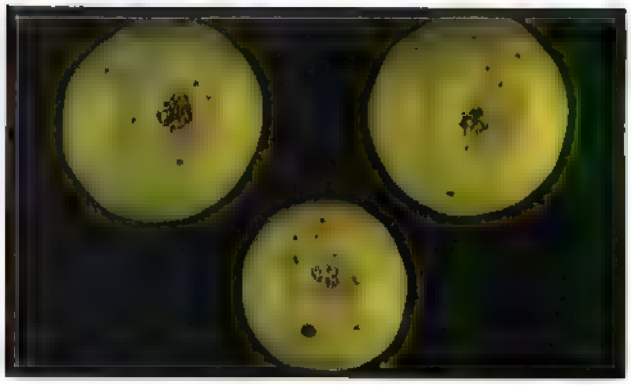

Fig. 43. Small black spots of black pox on Grimes apple.

spores from early bloom to about 3 wk after petal fall. During wet periods with suitable temperatures, the ascospores infect the blossoms and young fruit.

The disease is noted most often on Paulared, McIntosh, Rome Beauty and Delicious, although other varieties are undoubtedly infected.

Control

Blossom end rot has not caused sufficient damage to justify a study of control measures. Some of the fungicides used to control apple scab may help prevent infection.

In apples with higher levels of infection, prolonged storage may be undesirable due to secondary rot problems. This is particularly true of Delicious where blossom end rot often leads to moldy core problems.

HELPFUL REFERENCES

Abawi, G. S. and R. G. Grogan. 1975. Source of primary inoculum and effects of temperature and moisture on infection of beans by *Whetzelinia sclerotiorum*. Phytopathology 65:300-309.

Rich, A. E. 1970. Calyx-end rot of apples. Phytopathology 60:1152.

Black Pox

Black pox, caused by the fungus *Helminthosporium papulosum*, affects apple twigs, fruit and foliage. It is most severe in the southern Appalachian growing area but has been reported from most mid-Atlantic and lower mid-western states. The disease also affects pear.

Symptoms

On bark, infections begin as well-defined conical, shiny black elevations, called papules. Secondary growth of the fungus results in the formation of necrotic, dark, sunken areas 1/4 to 1/2 inch in diameter. Because bark becomes resistant with age, infections on older twigs are small and may not show any secondary enlargements. Symptoms of twigs infected with *H. papulosum* may be confused with internal bark necrosis, a physiological disorder.

Fruit infections appear as smooth, black, slightly sunken round spots, 1/8 to 3/8 inch in diameter (Fig. 43).

Leaf spots begin as small (1/16 inch) light green lesions surrounded with a red halo. Lesions enlarge to 1/16 to 1/4 inch in diameter, and the centers become tan to brown.

Pathogen Identification

Conidia can usually be produced at the margins of the papules or bark cankers if infected twigs are incubated in a moist chamber. Conidia are 29 to 45 by 6.6 to 8.2 μm with three or more septations. The fungus is easily isolated from fruit lesions onto culture media. Sporulation is poor in culture, but sufficient spores are usually produced to identify the fungus.

Disease Cycle

Helminthosporium papulosum overwinters in twig cankers. Conidia produced on these cankers are blown or washed onto leaves, twigs and fruit. Bark on twigs remains susceptible for several years. Rainy periods with temperatures of 70 to 80°F are most favorable for infection.

Control

Disease-free planting stock is important to help prevent the spread of the disease. Most fungicides used in the cover sprays at 14-day intervals for summer disease control satisfactorily control black pox.

HELPFUL REFERENCES

Berg, A. 1933. Black pox and other apple-bark diseases commonly known as measles. West. Va. Agr. Expt. Sta. Bull. 260. 31 pp.

Taylor, J. 1963. An apple leaf spot caused by *Helminthosporium papulosum*. Plant Dis. Rep. 47:1105-1106.

Brooks Spot

Brooks spot, caused by the fungus *Mycosphaerella pomi*, occurs in most of the apple-growing areas in the eastern United States. It is most severe in the southeastern mid-Atlantic, and northeastern apple-growing regions. Although considered a minor disease, losses greater than 50% occur in some orchards. The disease is also known as fruit spot or Phoma fruit spot. The disease occurs on quince and is called quince blotch.

Symptoms

Brooks spot first appears on immature fruit as irregular, slightly sunken, dark green lesions. As fruit ripen on red-skinned varieties, the lesions become dark red to purple to almost black. On light-skinned varieties, lesions remain dark green (Fig. 44). In these lesions, scattered pycnidia often develop beneath the epidermis. Lesions are generally more common on the calyx end. Fruit may crack if infections occur early and are numerous. Lesions may be confused with cork spot or bitter pit, but unlike cork or bitter pit, lesions are shallow, and the flesh of the fruit is not corky beneath the lesions. Jonathan, Stayman, Rome Beauty and Golden Delicious are susceptible Delicious is resistant.

Pathogen Identification

Mycosphaerella pomi is not readily identified without culturing on an agar medium. Although pycnidia of the imperfect or Phoma stage are formed on fruit, spores are seldom formed. The fungus is easily isolated from fruit lesions and forms the *Cylindrosporium* stage on culture media. Conidia are hyaline, and 2- to 5-celled, 2.0 to 2.5 by 15 to 60 μm.

Fig. 44. Brooks spot on apple fruit *(courtesy K.S. Yoder, Virginia Polytechnic Institute and State Univ.).*

Disease Cycle

Mycosphaerella pomi overwinters in apple leaves on the orchard floor. During the winter and spring, perithecia form, and ascospores are matured and discharged during rainy periods in May and June. Conditions favoring infection have not been precisely defined. Pycnidia are formed on infected fruit; however, spores produced in them are not thought to play a significant role in the life cycle.

Control

Most of the fungicides used in the early cover sprays for summer diseases adequately control Brooks spot.

HELPFUL REFERENCES

Brooks, C. and C. A. Black. 1912. Apple fruit spot and quince blotch. Phytopathology 2:63-72.

Blotch

Blotch, caused by the fungus *Phyllosticta solitaria*, was at one time considered second only to scab as the most important apple disease in the eastern United States from Pennsylvania, Indiana, and Ohio southward. Today, the disease rarely occurs in commercial apple plantings.

Symptoms

Blotch affects leaves, buds, twigs and fruit. Interveinal lesions on leaves begin as circular yellowish-green spots about 1/16 inch in diameter. A single pycnidium often forms in the center of each spot. As the spots age, they turn light tan to white. Spots on leaf veins and petioles are often much larger and may girdle the petiole and cause defoliation. Several hundred spots may occur on a single leaf.

Twig and branch cankers are often located at leaf nodes or at the base of spurs that have developed from dormant buds. Infections on current season's growth appear in late summer. Young cankers are dark, purplish or black and raised or blister-like. During the second season, cankers enlarge and become light tan or orange. Older cankers become roughened as they enlarge, and bark is sloughed off.

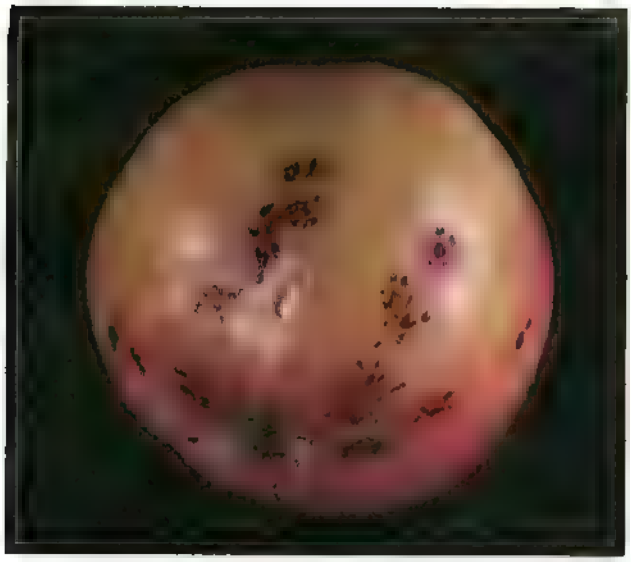

Fig. 45. Apple blotch on the fruit.

Fig. 46. X-spot on the calyx end of apple fruit *(courtesy K.S. Yoder, Virginia Polytechnic Institute and State Univ.).*

Cankers may coalesce, girdling twigs and small branches. Pycnidia are produced in cankers.

Fruit infections begin as small, dark, somewhat raised blister-like areas. Lesions enlarge slowly and may attain 1/2 inch or more in diameter. Lesions are slightly raised with irregular or lobed margins (Fig. 45). As lesions age, they turn from dark brown to black. Infected tissues harden and may crack. Pycnidia may be produced in the lesions.

Pathogen Identification

Pycnidia are produced in twig cankers, on leaf spots and in fruit lesions. Conidia are unicellular, ovoid, hyaline and 7 to 11 by 6 to 8.5 μm. They may be enveloped in a gelatinous sheath, which is frequently elongated and thread-like at the apical end.

Disease Cycle

The blotch fungus overwinters in twig and branch cankers or infected fruit. Conidia produced in pycnidia in the cankers are washed or blown onto fruit, foliage and twigs. Fruit infection can occur anytime from petal fall until harvest, although as fruit mature, they become more resistant. New leaf and twig growth, particularly watersprouts, are susceptible throughout the growing period. The optimum temperature for infection is 77 to 86°F. Old varieties such as Dutchess and Ben Davis, Black Twig and Yellow Transparent, are very susceptible. Delicious and Winesap are resistant. The planting of resistant varieties is one of the main reasons this disease is no longer important.

Control

Control is achieved through planting disease-free nursery stock, planting resistant varieties, such as Delicious, and following a regular spray program.

HELPFUL REFERENCES

Guba, E. F. 1924. Phyllosticta leaf spot, fruit spot, and canker of apple—its etiology and control. Phytopathology 14:234-237.

Kohl, E. J. 1932. Investigation on apple blotch. Phytopathology 22:349-369.

X-Spot

X-spot occurs in the southern Appalachian apple growing region. The causal organism has not been identified, although *Nigrospora oryzae* has been associated with the disease.

Symptoms and Disease Cycle

X-spot is characterized by small, circular, shallow, light tan necrotic spots which often occur at the calyx end of the fruit (Fig. 46). The disease is most common late in the season.

Factors affecting disease development are not known.

Control

Fungicides applied for "summer diseases" give effective control of X-spot.

Apple Mosaic

This virus disease was once very common in apple orchards because it was transmitted through the nursery stock. Nurserymen now use budwood free of this virus. Symptoms occur as creamy-yellow areas in leaves on one to a few branches of the tree (Fig. 47). This disease is found mainly in old orchards and is described here because the symptoms are sometimes confused with damage from herbicides.

Stony Pit of Pear

Stony pit, a virus disease, severely misshapes and gnarls pear fruit, making them unsaleable (Fig. 48). Less severely infected fruit may be dimpled, a symptom often confused with plant bug injury. Grit cells concentrate beneath pits and make it difficult to cut the fruit with a knife. A rather pronounced, roughened bark or "measles-like" symptom has been associated with some stony pit strains. Leaf symptoms are known but are difficult to distinguish in the orchard. Bosc is most severely affected. Anjou, Winter Nelis and Waite are affected to a lesser extent

Control

To prevent problems with this virus in new plantings, select trees known to be virus-free. While it is best to remove infected trees, removal of all infected Bosc trees in some orchards could reduce Bartlett pear yields due to lack of sufficient pollenizers.

HELPFUL REFERENCES

Kienholz, J. R. 1939. Stony pit, a transmissible disease of pears. Phytopathology 29:260-267.

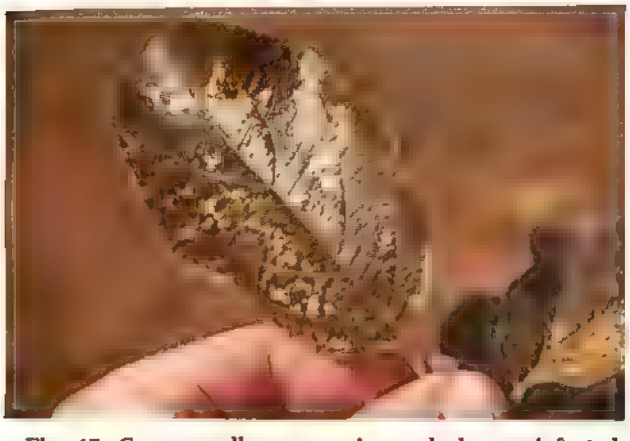

Fig. 47. Creamy-yellow areas in apple leaves infected with apple mosaic virus.

Fig. 48. Pear fruit severely gnarled from stony pit virus.

Nectria Twig Blight

Nectria twig blight is a minor disease that results in dieback of apple twigs. It is caused by the fungus *Nectria cinnabarina*, imperfect state of *Tubercularia vulgaris*. Recognition of the disease is important because it is often confused with fire blight, which requires different control measures.

Symptoms and Disease Cycle

In June, shoot growth on infected twigs begins to wilt and die. Small, sunken cankers are found at the base of the wilted shoots. Unlike fire blight, leaves on infected shoots appear to die from their base, not from the tip, and no signs of blighted blossom clusters remain on the twigs. In mid- to late-summer, bright orange or coral-red structures (sporodochia), 1/8 to 1/6 inch in diameter, appear on the surface of the cankers (Fig. 49). In autumn, the fungus has been reported to produce clusters of small, dark red, globular perithecia, but these structures have rarely been seen in the eastern United States.

Optimum fungus growth occurs in culture at 70°F, moderate growth at 80 to 85°F, and limited growth at 37°F. Picking wounds, which probably take longer than usual to heal in late-maturing seasons, combined with prolonged periods of wet weather after harvest, appear to favor the establishment of infection. The disease has been noted primarily on varieties with a large cluster-bud base such as Rome Beauty, Ben Davis and Northern Spy.

Control

The disease is not usually severe enough to require special control measures. As a result, chemical control procedures have not been developed. Removal of infected twigs helps reduce the carry-over of inoculum.

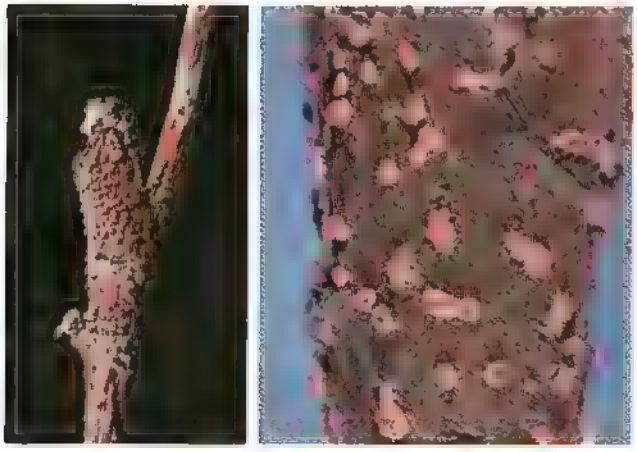

Fig. 49. Bright orange fruiting structures of Nectria twig blight fungus (enlarged at right).

Fig. 50. Blossom blast of pear. Note death of spur but failure of shoot to become infected.

HELPFUL REFERENCES

Jones, A. L. 1963. Occurrence of *Nectria* twig blight of apple in western New York in 1962. Plant Dis. Rep. 47:538-540.

Thomas, H. E. and A. B. Burrell. 1929. A twig canker of apple caused by *Nectria cinnabarina*. Phytopathology 19 1125-1128.

Blossom Blast of Apple and Pear

Blossom blast is caused by the bacterium *Pseudomonas syringae* pv. *syringae*. The disease has been reported from several northeast and north central states. Although the disease is very sporadic, it may be very destructive when it occurs.

Symptoms and Disease Cycle

During cold, rainy periods around the bloom period, blossoms of apple and pear sometimes are infected. The disease causes a blighting of the blossoms and young leaves. Symptoms are often restricted to the lower part of the tree or to trees in low-lying portions of the orchard. The disease is referred to as blossom blast because death is sudden and very young buds and flowers are killed.

Symptoms of bacterial blast differ from those of fire blight in that bacterial ooze, a common characteristic of fire blight, is not found on the infected tissues. Also, blast infections rarely extend to the shoot growth (Fig. 50). Immature fruit, particularly pear, will sometimes develop water-soaked areas that become firm black lesions.

For information on the isolation and identification of this pathogen, see blister spot of apple and bacterial canker of stone fruits.

Control

Effective control measures are lacking. Streptomycin spray applications starting early in the bloom period have shown promise on pears in Connecticut.

HELPFUL REFERENCES

Parker, K. G. and W. H. Burkholder. 1950. *Pseudomonas syringae* Van Hall on apple and pear in New York State. Plant Dis. Rep. 34:100-101.

Sands, D. C. and J. L. McIntyre. 1977. Possible methods to control pear blast, caused by *Pseudomonas syringae*. Plant Dis. Rep. 61:311-312

Thread Blight

Thread blight is caused by the fungus *Ceratobasidium stevensii* (*Pellicularia koleroga* and *Corticium stevensii* in some literature). The disease is primarily a problem on apple in the southeastern United States. In poorly managed orchards, some apple growers in the southern Appalachian region have suffered severe losses.

Symptoms and Disease Cycle

Thread blight symptoms are readily noticed in early summer as leaves wilt and turn brown, usually in the interior or shaded portions of the tree (Fig. 51). Dead, curled leaves cling to blighted branches, frequently in mid-branch with unaffected leaves still appearing distal and proximal to the diseased area. In well-managed orchards, the disease is usually not seen until after harvest, when the fungicidal spray program has been discontinued.

Fig. 51. An apple branch affected with thread blight showing dead leaves still attached.

Positive diagnosis in the field is made by observing signs of the fungus. A sparse, white mycelial fan can be observed in advance of dead areas on partial blighted leaves. This mycelium can frequently be traced as fine white threads back to the leaf petiole and twigs. Abscised leaves may be tied to twigs and leaves by this network of threads and mycelium. The fungus is present on twigs and branches as silvery-tan rhizomorphs and white to tan sclerotia, which become hard and dark brown with age. Rhizomorphs may be up to 1/16 inch wide and sclerotia up to 3/16 inch long and 1/8 inch thick. These fungal structures are superficial and can be scraped from the bark. The bark and wood of blighted branches do not appear adversely affected by the disease. Sclerotia and rhizomorphs can also grow superficially on fruit.

Information on the disease cycle of thread blight is limited. Sclerotia retain viability over the winter under eastern Kentucky conditions. Leaf blight usually appears first in the late spring on branches having sclerotia from the previous season. During the season, the fungus may grow from blighted to adjacent healthy leaves. The disease is more severe when trees are growing in a moist, shaded environment.

Control

Thread blight, once established in an orchard, is difficult to control using fungicides. Preventative fungicides applied to trees prior to infection may protect the orchard from thread blight. Selection of the orchard site, avoiding "hollows" and other shaded and poorly ventilated areas should help to prevent the disease. Under light disease pressure, pruning blighted twigs and branches may provide adequate control; pruning to promote better penetration of sunlight and air may also help.

Necrotic Leaf Blotch

Unlike the other diseases described in this bulletin, the cause of necrotic leaf blotch (NLB) is not known. It is suspected to be a physiological disorder. The disease occurs on Golden Delicious and its bud sports and was first reported in the United States in 1969. It has been studied primarily in the southeastern United States but occurs wherever Golden Delicious is grown. NLB occurs periodically throughout the eastern fruit growing areas.

Symptoms and Disease Development

The disease is characterized by the development of necrotic blotches or irregular areas of dead tissue in mature leaves (Fig. 52). Midshoot leaves are most often affected. Blotches are limited by the leaf veins and vary greatly in size. Affected leaves begin to turn yellow after about 4 days and abscise a few days later. Some green leaves with NLB are also lost by abscission.

Two distinctive characteristics of NLB are that the symptoms develop suddenly, almost overnight, and in waves, generally from June through August. However, the disease tends to be more common and severe later in the summer. Some orchards or trees within an orchard may show little or no defoliation, while other orchards or trees reach 50% defoliation or more (Fig. 53).

Control

Although it appears NLB is not caused by a fungus, bacterium or certain air pollutants, the disease is reduced where certain ethylene-bis-dithiocarbamate fungicides are used in the spray schedule to control summer diseases.

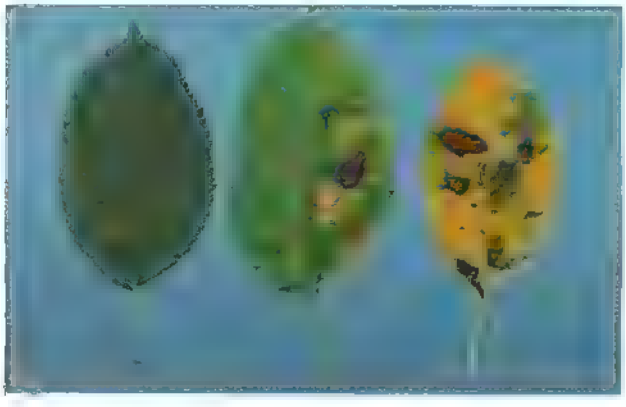

Fig. 52. Golden Delicious leaves with necrotic leaf blotch. Early symptom development is shown in leaf on left; greatly advanced stage on right.

Fig. 53. Leaf yellowing and defoliation of Golden Delicious from necrotic leaf blotch.

Soft Rot/Blue Mold

Soft rot or blue mold is the most common storage rot of apples. It is caused primarily by the fungus *Penicillium expansum*, but other *Penicillium* spp. produce a similar rot. It occurs on apple, pear and numerous other crops.

Symptoms and Disease Cycle

Soft rot can usually be distinguished by its light color and the soft, watery texture of the decayed areas. The rot develops rapidly under favorable temperatures, often rotting the whole fruit in about 2 wk. When humidity is high, the fungus develops gray-blue, cushion-like structures on the surface of the rot (Fig. 54). These structures produce spores (conidia) important in spread of the disease (Fig. 55).

Spores of the soft rot organism are extremely resistant to drying and can survive on surfaces of packing and picking equipment. Thus, spores are nearly always present to cause trouble when conditions favor infection and disease development. Spores often build up in water used in dumping bulk boxes of fruit or in solutions used to treat apples for scald.

Soft rot is a disease of ripe fruit, which develops mostly on apples picked at an advanced state of maturity. It rarely occurs on immature fruit or in the orchard, except on fallen fruit. The pathogen infects the fruit through skin breaks and lenticels. Although stem punctures are usually considered the most important point of entrance, infection through lenticels in bruised areas may be serious at times.

Control

To reduce soft rot, harvest fruit at optimum maturity, handle carefully to prevent bruises and other injuries, and put into cold storage as soon as possible after harvest. If fruit are to be stored late into the season, use disinfectants or fungicides in the water during grading to reduce spread to healthy fruit. In some areas, strains of *P. expansum* have developed resistance to benomyl. Contact your local extension agent for suggested chemical controls.

Fig. 54. Golden Delicious apple with soft rot (top). Note cushions of blue-green fungus growth on surface lesion. McIntosh with gray mold (bottom). Note breakdown of the fruit.

HELPFUL REFERENCES

Pierson, C. F., M. J. Ceponis, and L. P. McColloch. 1971. Market diseases of apples, pears, and quinces. U.S. Dept. Agr., Handbook 376. 112 pp.

Gray Mold

Gray mold of apple and pear, caused by *Botrytis* spp., is second in importance to soft rot as a decay or rot problem of fruit in cold storage. When fruit are removed from storage after several months, gray

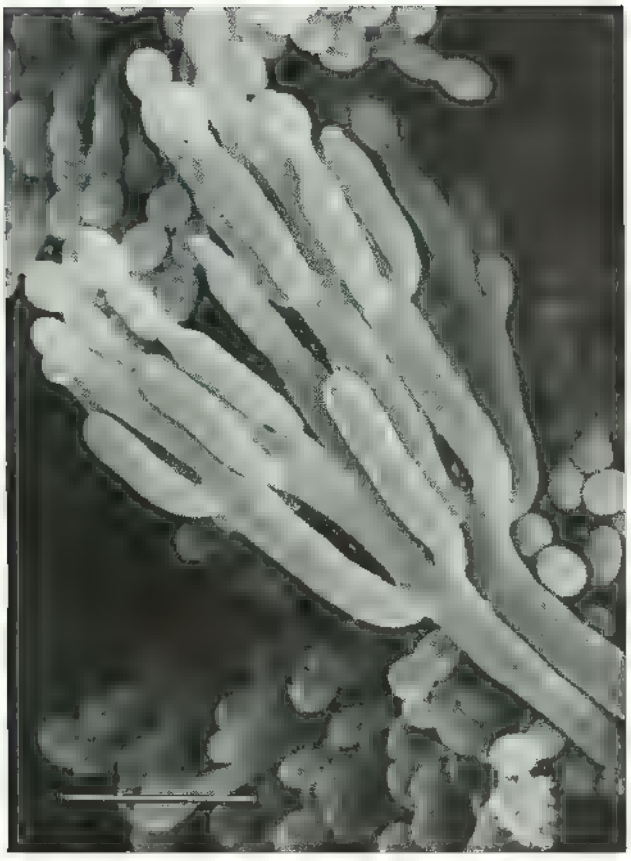

Fig. 55. Conidiophores of the soft rot pathogen *Penicillium expansum*. Note one-celled ovoid conidia produced on brush-like structures.

mold infected fruit will be almost completely decayed (Fig. 54). Often, infection spreads from fruit to fruit, producing "nests" or "pockets" of decayed fruit. Although gray mold infection may occur on individual fruit in the orchard and develop in storage, spores of the fungus often build up in solutions used to treat apples for scald or internal breakdown, and in water used in dumping bulk boxes. Addition of an effective fungicide to these solutions will prevent spread of the disease.

Bull's-Eye Rot

Bull's-eye rot, or "northwestern anthracnose," is primarily a problem on fruit in storage, although infection originates in the orchard. It is a minor disease in the fruit growing areas of the eastern United States. The fungus causing the disease is *Neofabraea malicorticis*.

Symptoms

Small, elliptical, sunken cankers develop in the bark, on twigs and branches that are less than 2 inches in diameter. Old cankers have a pronounced ridge of callus around the margin. New infections appear in the autumn as small reddish-brown areas on young twigs. As the cankers enlarge during the autumn and spring, the bark remains smooth. Later cracks develop between the canker and surrounding healthy tissue.

Infected fruit develop brown, sunken, round lesions on the surface (Fig. 56). The centers of the infected areas are often light colored and surrounded by alternating areas of tan and brown, giving the characteristic "bull's eye" appearance. Lesions tend to be relatively small. Whole fruit are rarely rotted by one lesion. The surface of the rot is sometimes covered with cream-colored spore masses.

Disease Cycle

Conidia of the bull's-eye rot fungus are washed by rain from cankers on the twigs and branches to the apples and twigs below. Ascospores formed in 2-yr-old cankers are forcibly discharged and disseminated by wind. Infection is mainly through the lenticels on the fruit, but wounds may also be infected. Fruit decay does not usually show up until after harvest.

The disease is generally worse in years with frequent rains and moderate temperatures during the autumn and on fruit harvested beyond optimum maturity. Twig infections develop over a 2-yr period. In the first autumn after infection, conidia are produced in acervuli on the surface of the canker; ascospores are produced in apothecia borne on cankers the second year.

Control

Removal of twigs with cankers aids control by reducing the number of fungal spores. Chemical sprays near harvest help control infection to the fruit and twigs.

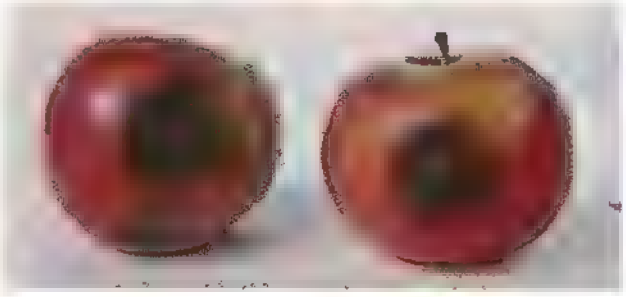

Fig. 56. Bull's-eye rot. Note light color in center of apple on right and alternating areas of tan and brown on fruit at left.

Moldy Core

Moldy core is primarily a problem on fruit of Delicious. The problem originates in the orchard, and infected fruit sometimes color and ripen prematurely. Usually infected fruit appear normal when harvested and are not detected until prolonged storage. When infected fruit are cut in half, the seed cavity, or core region, is overgrown with mold (Fig. 57). A small proportion of the fruit may develop some rot in the flesh after prolonged storage or after the fruit are sold and held at room temperature.

Many genera of fungi can be isolated from the core region of apple. *Alternaria* spp. are the most common. Presumably, infection occurs through calyx tubes that remain open and allow spores to enter the seed cavity, where conditions are excellent for fungal development.

Control

There are no control measures. The incidence of moldy core has not been reduced by common fungicide spray programs, probably because once a fungus reaches the core region it is protected against contact with fungicides.

HELPFUL REFERENCES

Carpenter, J. B. 1942. Moldy core of apples in Wisconsin. Phytopathology 32:896-900.

Ellis, M. A. and J. G. Barrat. 1983. Colonization of Delicious apple fruits by *Alternaria* spp. and effect of fungicide sprays on moldy-core. Plant Dis. 67:150-152

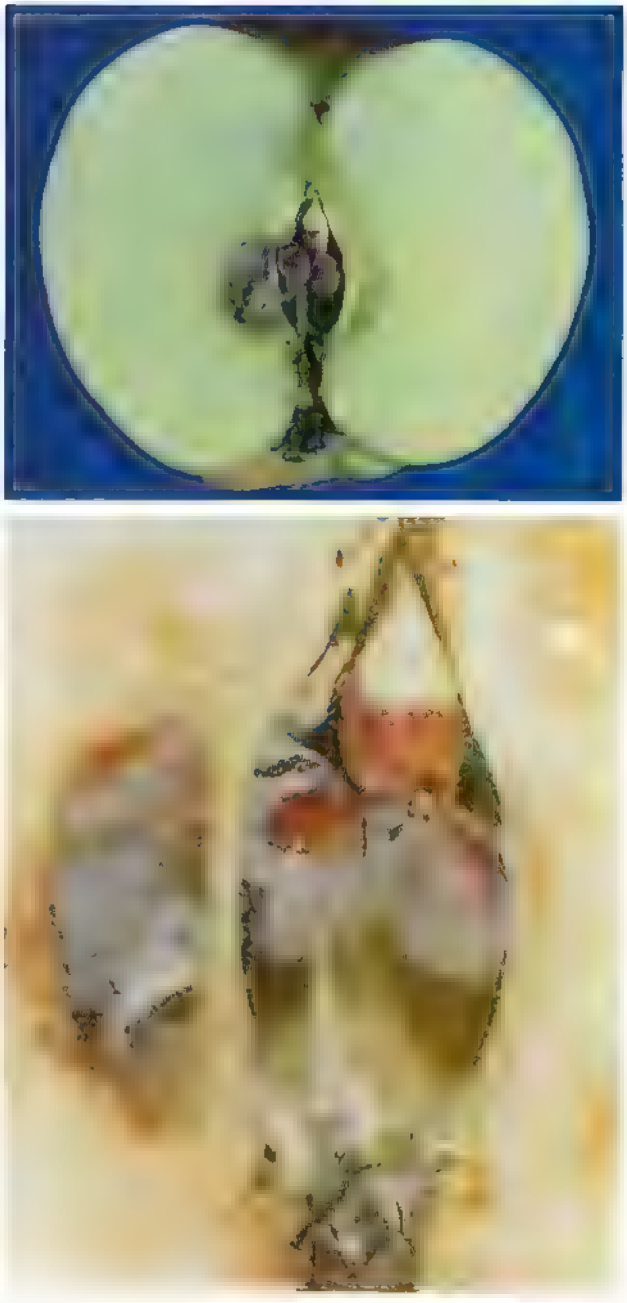

Fig. 57. Cross section of apple with moldy core developing around the core region (top) and close-up of seed cavity overgrown with mold (bottom) *(courtesy M.A. Ellis, Ohio Agricultural Research and Development Center, Wooster).*

Phytophthora Collar Rot

Collar rot is caused by the fungus *Phytophthora cactorum*. Although *P. cactorum* is most often associated with the disease, *P. cambivora* and other *Phytophthora* spp. may also be involved. The disease was a minor problem when apple trees were propagated on seedling rootstock except where Grimes apples were grown. The incidence of *Phytophthora* collar rot has increased as size-controlling rootstocks have replaced seedling rootstocks.

Symptoms

The disease produces cankers at or below the ground line in the root-crown area (Fig. 58). Infected bark becomes brown and is often soft and mushy or slimy when wet. Cankers may extend from the point of origin into the root system and up the trunk to the bud union. If the scion variety is susceptible to *Phytophthora*, the disease may extend above the union. More often, the disease stops at the ground line. Older cankers form a definite outline when the bark dries out, and callus tissues may develop at the margins.

The cankers girdle the roots and lower trunk, resulting in poor terminal growth and foliage discoloration. Severely infected trees eventually die.

Fig. 58. Phytophthora collar rot. In later stages, the diseased area is completely killed, resulting in death of the tree.

Fig. 59. Sporangiophores of *Phytophthora cactorum* in which large numbers of motile zoospores are produced.

The leaves on affected trees may be small and yellow in summer or turn reddish to purple in early autumn. Because other root problems cause similar symptoms, these symptoms only indicate the need for further examination of the root system. Often, isolation of the fungus is required before a positive diagnosis can be made.

Young trees show the disease most when fruit production begins. This could be in the third year on some early-bearing rootstocks. Infected trees usually linger 2 or 3 yr before dying. However, if conditions are optimum for infection, trees can be killed in one growing season.

Disease Cycle

The pathogen survives in soil for several years as oospores. These thick-walled structures can resist periods of unfavorable environment, such as drought, and are relatively resistant to chemical treatment. They occur in greatest numbers in old orchard soils. The fungus is also believed to overwinter as mycelium in diseased tissue.

Fungal growth and infection are favored by damp, cool periods after harvest and in spring about the time leaves and flowers are emerging. The ability to produce many spores (primarily zoospores) allows the fungus to build up to high levels from a few oospores under favorable conditions (Fig. 59). Zoospores swim in films of moisture to the crown area where they infect. Others may be splashed on the fruit and cause a rot. Rotten fruit are firm and light tan. *Phytophthora* fruit rot is most common in areas where the water used for overhead irrigation contains zoospores from infected forest or fruit trees.

Control

Collar rot can be reduced by carefully selecting the orchard site and rootstocks for the new planting. Susceptible rootstocks should not be planted in orchards (or areas of the orchard) with heavy, poorly drained soils. Tiling of wet areas in an otherwise well-drained location often improves internal soil drainage sufficiently to avoid the disease in low areas.

Planting on a raised soil bed greatly improves drainage around the tree and can reduce collar rot incidence. Where the soil is variable, it becomes necessary to use several different rootstocks in the orchard, depending on their soil preference and susceptibility to collar rot.

The clonal rootstocks MM 104, MM 106 and M.26 are very susceptible. M. 7 and M. 2 are considered susceptible, but extensive use of these stocks in the eastern United States indicates they have some field resistance. Malling 9 is considered resistant. Seedling rootstocks of Golden Delicious, Delicious and Rome Beauty are moderately resistant, while those of Melba, McIntosh and Wealthy are apparently resistant. Tests and observations on other rootstocks are limited. Disease-free nursery stock should be planted, since infected nursery stock has been shown

to be an important source of the disease.

Once infection occurs, it is difficult to eradicate the disease and save the tree. Probably the most effective method is the use of "nurse trees." This practice involves inarching 1-yr-old whips of a resistant variety into the trunk above the lesion. Results are best when treatments are made early but are of limited value if more than 50% of the circumference of the trunk is infected.

Chemical control procedures are being tested. At best, they are "stop-gap" measures for use in emergencies; they are not a substitute for selecting rootstocks adapted to the intended orchard site.

HELPFUL REFERENCES

Baines, R. C. 1939. Phytophthora trunk canker or collar rot of apple trees. Journal of Agr. Res. 59:159-184.

Jeffers, S. N., H. S. Aldwinckle, T. J. Burr, and P. A. Arneson. 1982. *Phytophthora* and *Pythium* species associated with crown rot in New York apple orchards. Phytopathology 72:533-538.

Mircetich, S. M. and M. E. Matheron. 1976. Phytophthora root and crown rot of cherry trees. Phytopathology 66:549-558.

Armillaria Root Rot

This disease occurs primarily on peach and cherry but is an occasional problem on apple and rare on pear. Infection of apple, grown on seedling rootstock, is usually restricted to a few trees in an orchard. Susceptibility of clonal apple rootstocks to infection is not known. Details of this disease are covered in the stone fruit section of this bulletin.

Clitocybe Root Rot

This disease occurs primarily in the southern portion of the United States and is more common on peach than on apple. Root infections resemble those of Armillaria root rot and may be confused with it if mushrooms or rhizomorphs are not present. For details of this disease, see the stone fruit section of this bulletin.

White Root Rot

White root rot is caused by the fungus *Corticium galactinum*. The disease occurs from Canada to Texas, although is it most prevalent in the southeastern apple-growing areas. Trees affected with white root rot usually die more rapidly than those affected with other root rots.

Symptoms and Disease Cycle

The surface of affected roots is covered with white or cream-colored mycelium (Fig. 10). The mycelial growth is often thick and may extend into crevices in the soil close to roots. The cambium of affected roots is not always uniformly killed; as a result, zonate spots may be visible in the wood when the bark is removed. Affected roots are soft and light in weight.

Many forest trees and shrubs are hosts for *C. galactinum*. White oak is one of the most important hosts. Infection occurs when apple roots contact infected root pieces in the soil. The mycelium may remain viable on infected root pieces in the soil for many years.

Control

No practical control is known. Stumps and roots of forest trees should be thoroughly removed from recently cleared sites. Replanting infested sites in established orchards is often not successful. If replanting is attempted, removal of all old roots is essential. No resistant rootstocks are available.

HELPFUL REFERENCES

Cooley, J. S. and R. W. Davidson. 1940. A white root rot of apple trees caused by *Corticium galactinum*. Phytopathology 30:139-148.

Fig. 60. White mycelium of the white root rot fungus growing over the surface of the trunk.

Black Root Rot

Black root rot of apple is caused by the fungus *Xylaria mali*. It is primarily important in the southern Appalachian states. *Xylaria* has also been reported to cause a root rot of pear and sweet cherry. Peach is not affected.

Symptoms and Disease Cycle

The above-ground symptoms of trees affected with black root rot are similar to those caused by other root rot pathogens, vole injury, etc. Affected roots are brittle, easily broken and covered with a black fungal encrustation. Decayed wood is light, dry and the bark remains firmly attached to the root. Trees with advanced root rot are easily uprooted. Finger-like fruiting structures are occasionally found above ground at the base of affected trees (Fig. 61). These are initially white but turn black as they mature.

The life cycle of *X. mali* is not clear. Only a few wild hosts have been reported, although *Xylaria* spp. are saprophytes on many plant species. Mycelium in dead wood in the soil is the most important inoculum source. The fungus can survive in infected roots for 10 yr or longer. The role of ascospores and conidia in the disease cycle is unknown.

Control

Satisfactory controls are not available. Sites known to be infested with *Xylaria* should not be planted. If replanting is attempted, all old roots should be carefully removed. Fumigation of the tree site with methyl bromide may be helpful. There are some differences in rootstock susceptibility although none are resistant. MM 104 and MM 111 are more susceptible than MM 106 and seedling rootstock.

HELPFUL REFERENCES

Cooley, J. S. 1944. Some host-parasite relations in the black root rot of apple trees. Journal of Agr. Res. 69: 449-458.

Fromme, F. D. 1928. The black root rot disease of apple. Va. Agr. Expt. Sta. Tech. Bull. 34. 52 pp.

Southern Stem Blight

Southern stem blight, caused by the fungus *Sclerotium rolfsii*, is primarily a problem in the Piedmont apple-growing areas in the southeastern United States. *Sclerotium rolfsii* is a widespread pathogen, and affects several hundred plant species.

Symptoms and Disease Cycle

Sclerotium rolfsii affects the lower stem and roots of apple trees, killing the bark and girdling the trees (Fig. 62). The disease is characterized by a white web-like mycelium which often forms at the base and on the lower stem of affected trees (Fig. 63). Light brown to yellow, round sclerotia, 1/16 to 1/8 inch in diameter, form in the mycelial mat. The fungus is spread by the sclerotia, which also serve as overwintering structures. The disease is most severe on 1-to 3-yr-old trees. As the bark thickens, trees become resistant to infection.

Control

Avoid planting sites where the disease has been severe on previous crops such as peanut, clover, tomato and soybean. Keep the soil around the base of the tree free of dead organic matter, since it may serve as a food base for *S. rolfsii*. Some differences exist in rootstock susceptibility. The most resistant

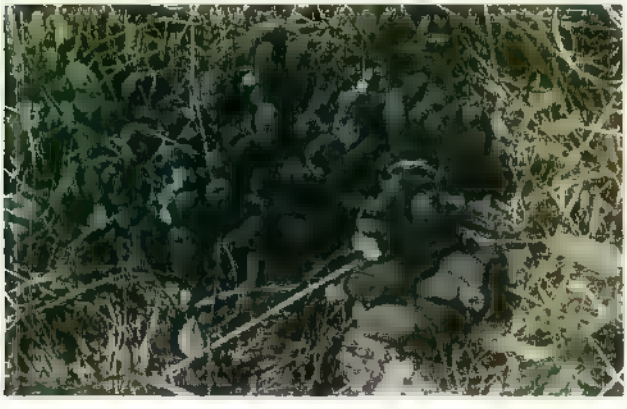

Fig. 61. Finger-like fruiting structures of the black root rot pathogen *Xylaria mali*.

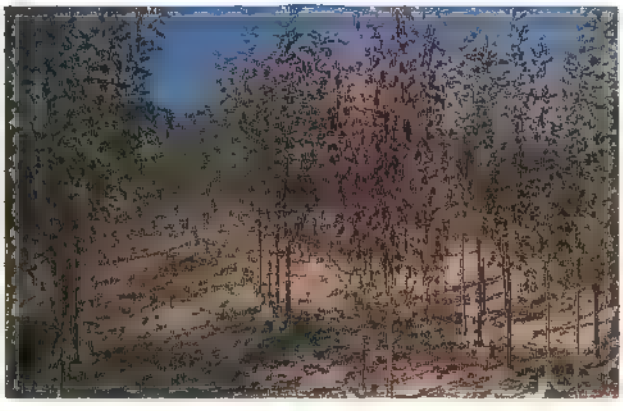

Fig. 62. Trees in a nursery row with dieback due to infection at soil line by the Southern stem blight pathogen.

Fig. 63. White, web-like mycelium of *Sclerotium rolfsii*. Light brown to yellow, round sclerotia often form in the mycelial mat.

Fig. 64. Typical weak growth and pale coloration of foliage of Delicious on MM106 rootstock with apple union necrosis and decline.

Fig. 65. Line of dead tissue at the union between the Delicious scion and the MM106 rootstock. Typical of union necrosis and decline.

rootstock currently used is M. 9. No fungicides are currently registered on apples for southern stem blight control.

Apple Union Necrosis and Decline

This disease was first reported on apple in Pennsylvania and Virginia, but subsequent observations and reports indicate it occurs in most northern apple growing areas. Certain clones of Delicious on MM 106 rootstock are the most severely affected.

Diseased trees exhibit general symptoms of decline, including small, sparse, pale leaves; reduced terminal growth, and early discoloration of the foliage (Fig. 64). Abnormally thick, spongy bark is found at the graft union, and when removed, a distinct line of necrotic tissue may be observed at the graft union (Fig. 65). Frequently affected trees produce large numbers of sprouts from the rootstock, and sometimes the top breaks off at the union in strong winds (Fig. 66).

Considerable circumstantial evidence indicates this disease is caused by the same virus that causes Prunus stem pitting, but direct transmission studies to prove this have not been completed. For further information on this disease, including its control, see Prunus stem pitting in the stone fruit section of this bulletin.

HELPFUL REFERENCES

Rosenberger, D. A., M. B. Harrison, and D. Gonsalves 1983. Incidence of apple union necrosis and decline, tomato ringspot virus, and *Xiphinema* vector species in Hudson Valley orchards. Plant Dis. 67:356-360.

Stouffer, R. F., K. D. Hickey, and M. F. Welsh. 1977. Apple union necrosis and decline. Plant Dis. Rep. 61.20-24.

Fig. 66. Breaking off of apple tree at the union between rootstock and scion—typical of union necrosis and decline.

Stone Fruits

American Brown Rot

Brown rot, caused by the fungus *Monilinia fructicola*, is a common disease of apricot, peach, nectarine, plum and cherry. It reduces yields by killing blossoms and twigs, and by rotting the fruit both on the tree and after harvest. In seasons when weather conditions are unusually favorable for infection, entire crops may be lost, almost overnight.

Symptoms

Brown rot attacks blossoms, spurs, shoots and fruit. Symptoms may develop on a few or all of these plant parts during the growing season. Infected blossoms wilt, turn brown, and persist into summer. Blighting of spurs from blossom infection is not common with American brown rot.

Fruit decay is more common on mature fruit than on immature fruit. Initially, small, circular, light brown spots develop on the surface of the fruit and expand rapidly under favorable conditions, destroying entire fruit in a few hours (Figs. 67-69). Rotted fruit may fall to the ground or persist as mummies on the tree.

The fungus often moves from infected fruit into spurs and small branches. Small cankers are formed that girdle the branch or spur and cause the terminal growth to wither and die. Gummosis often accompanies the blighting of spurs and formation of cankers. Succulent shoots are sometimes blighted by direct infection near their tip.

Under wet and humid conditions, ash-gray tufts of fungus develop over the surface of the lesions. These structures, called sporodochia, produce conidia important in spread of the disease. Appearance of conidia on lesions is the most obvious characteristic of brown rot.

Fig. 67. Decay of peach fruit from brown rot.

Disease Cycle

Brown rot overwinters in mummies on the tree or ground and in cankers (Figs. 70, 71). While fruit buds are opening, small, cup-shaped mushrooms called apothecia arise from mummies on the ground

Fig. 68. Sweet cherries infected with brown rot. Similar symptoms are produced on sour cherries.

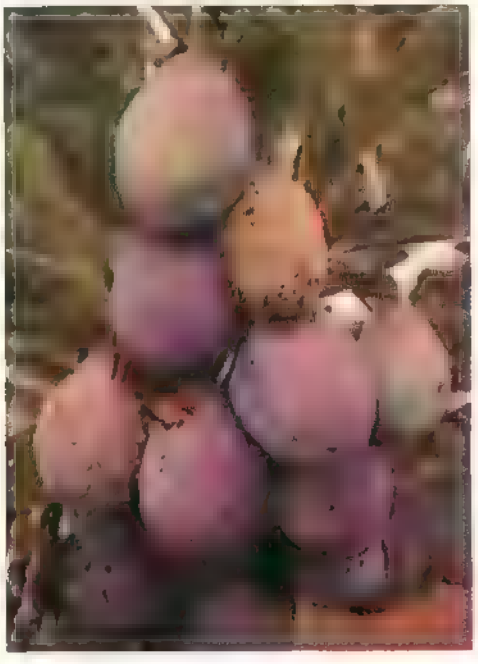

Fig. 69. Decay of Stanley prune and sporulation of the brown rot fungus.

(Fig. 72). These structures do not develop every year, as their development requires exact weather conditions. They are also hard to see among the debris on the orchard floor.

Spore-containing structures called asci, each with eight ascospores, are produced within the apothecia. Upon wetting, the ascospores are forcibly ejected into the air and carried by wind to blossoms where they infect. Apothecia disintegrate soon after bloom and do not contribute directly to infection at harvest.

Infection also arises from conidia produced on the surface of mummies and cankers in the tree. Spores are carried by wind or splashing rain to susceptible tissues. Relative humidity of 85% or higher is necessary for conidial production.

In summer, brown rot activity increases as the fruit start to mature. Infected blossoms and green fruit are the main source of conidia for infecting fruit at harvest. Infection may occur directly through the cuticle or through natural openings in the fruit. Wounded fruit are infected much more readily than non-wounded fruit. Since decay of the fruit and spore production can occur in a few days, the disease is able to build up rapidly.

Environment plays a key role in the development of the disease. Warm, wet, humid weather is particularly favorable for brown rot. The hours of wetting necessary for blossom infection decreases from 18 hr at 50°F to 5 hr at 77°F. Infection occurs slower above 80° and below 55°, but may continue at temperatures as low as 40°F. Mature fruit can decay in 2 days under optimum conditions for disease development.

Fig. 70. Brown rot mummy and cankered twig on peach at bloom.

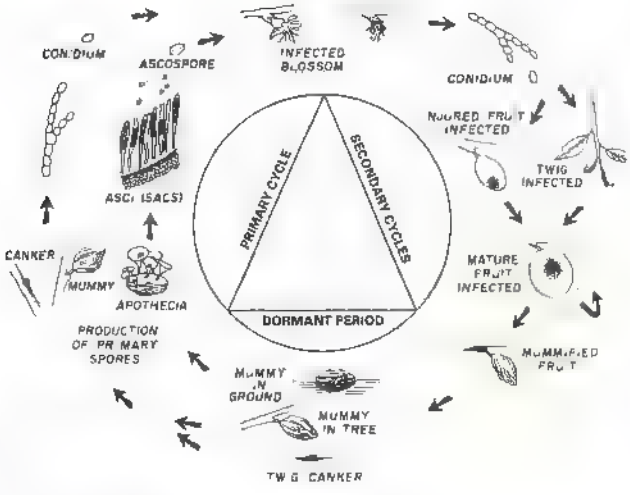

Fig. 71. Disease cycle of brown rot.

Control

The entire brown rot control program, although initiated before bloom, is designated to reduce decay of mature fruit. Several control measures help to prevent disease buildup at harvest.

Control starts with removal of all fruit, mummies and blighted twigs from trees after the last picking. This reduces the amount of brown rot overwintering in mummies and twig cankers. Trashy cultivation, just before bloom and no later than mid-bloom, will destroy the apothecia by disturbing the mummies.

Spray applications: Bloom sprays are an important consideration in the overall control program. Timing of sprays is based on development of the blossom buds and climatic conditions. The pistil is the most susceptible flower part to American brown rot. During wet weather, the first spray should be applied when many of the pistil tips (stigmas) extend above the flower, even unopened flowers. The number of sprays required during bloom varies from year to year. Spray applications every 4 to 5 days are necessary if favorable conditions for brown rot persist.

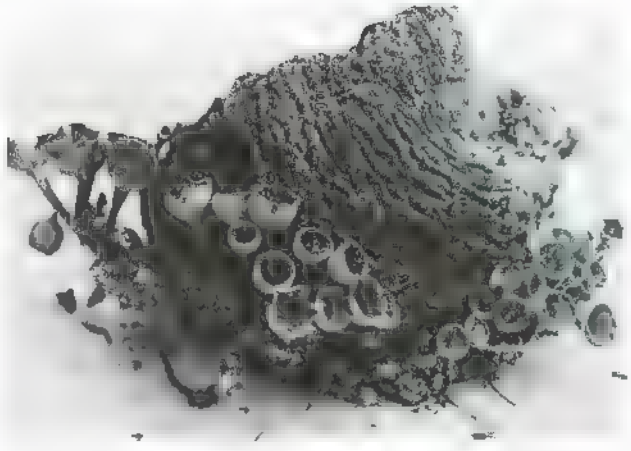

Fig. 72. Apothecia or cup-shaped fruiting bodies of brown rot arising from a peach mummy.

Pre-harvest sprays: To control brown rot on ripening fruit, apply pre-harvest fungicide treatments. Initiate sprays 3 wk before harvest or when fruit begins to color. Where brown rot has built up, the schedule should be initiated earlier and additional sprays or dusts made at closer intervals. Control of plum curculio, tarnished plant bug, and oriental fruit moth is essential to prevent early infection to immature fruit.

Post-harvest control: To reduce losses from decay during storage and in transit, pick and handle the fruit carefully. Use only clean containers and remove ripe and rotting fruit from packing areas periodically. Remove field heat from the fruit promptly after harvest by hydrocooling or forced air cooling. Other techniques that reduce decay are: hot water treatments, fungicidal dips, wax-fungicide treatments, and spraying the fruit while grading.

HELPFUL REFERENCES

Byrde, R. S. W. and H. S. Willetts. 1977. The brown rot fungi of fruit. Their biology and control. Pergamon Press. 171 pp.

Weaver, L. O. 1950. Effect of temperature and relative humidity on occurrence of blossom blight of stone fruit. Phytopathology. 40:1136-1151.

European Brown Rot

European brown rot, caused by *Monilinia laxa*, occurs primarily on sour cherry in New York, Michigan and Wisconsin. It is rare compared with American Brown rot. This disease is generally a blossom and spur blight, not a fruit rot (Fig. 73). Twigs are killed back up to 12 inches from blossom infections (Fig. 73). Conidia are produced on the dead spurs, twigs and cankers. The fungus overwinters in cankers, and spores are produced the following spring around the bloom period if adequate moisture is present. The main method of control is bloom sprays with effective fungicides. Pruning out infected spurs and branches also helps control the disease.

Fig. 73. Spur dieback of Montmorency sour cherries from infection by the European brown rot fungus.

HELPFUL REFERENCES

Calavan, E. C. and G. W. Keitt. 1948. Blossom and spur blight (*Sclerotinia laxa*) of sour cherry. Phytopathology 38.857-882.

Kable, P. F. and K. G. Parker. 1963. The occurrence of the imperfect stage of *Monilinia laxa* on *Prunus cerasus* var. *austera* in New York state. Plant Dis. Rep. 47:1104.

Cherry Leaf Spot

Cherry leaf spot, caused by the fungus *Coccomyces hiemalis*, reduces flower bud survival and decreases fruit set. The disease is common on sour and sweet cherries in the Great Lakes and mid-Atlantic states. A closely related species or "form" of the pathogen (*C. prunophorae*) is occasionally a problem on prunes. Although the disease is sometimes referred to as "shot-hole", cherry leaf spot is the preferred name.

Fig. 74. Spotting and yellowing of sweet cherry leaves due to infection by the cherry leaf spot pathogen.

Symptoms

The disease appears first on the upper side of the leaf as small, circular, purple spots. Later, the spots turn brown and form a definite boundary. On sweet cherry leaves, the spots are often larger and more nearly circular than those on sour cherry (Fig. 74). During rainy weather, light-pink to white masses appear on the underside of the leaf in the center of the spots (Fig. 75).

After the cherry leaves become infected, they turn yellow and fall. Only a few lesions per leaf can result in leaf yellowing and defoliation. The fruit on trees severely defoliated by leaf spot fail to mature properly, are light colored, low in soluble solids, and are soft and watery. They may be smaller or larger than normal, depending on when defoliation occurs and its severity. Early loss of leaves reduces tree vigor. The buds and wood are more susceptible to injury from low temperatures in winter.

Although the disease occurs primarily on the leaves, fruit, stems and leaf petioles may become infected when the disease is severe. Fruit infection is sometimes so severe that the crop is unmarketable.

Disease Cycle

The fungus overwinters in infected leaves on the orchard floor (Fig. 76). In late winter and early spring, it produces fruiting bodies (apothecia, Fig. 77) which normally mature during the blossoming period. Upon wetting, the ascospores are released. Greatest ascospore discharge occurs at temperatures of 61°F and higher, somewhat less at 54°F, and very little from 39 to 46°F.

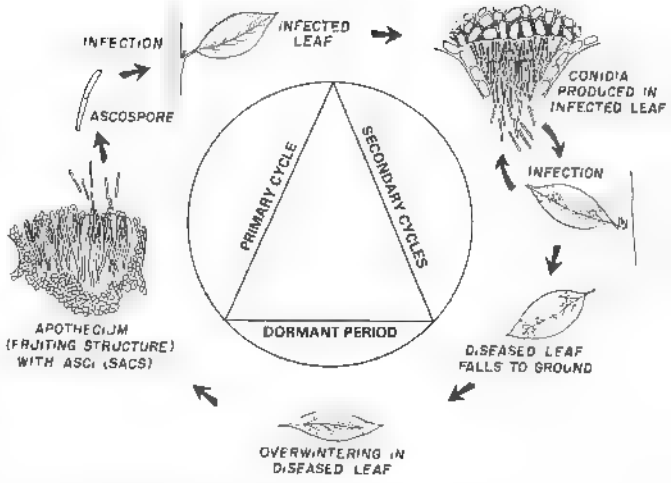

Fig. 76. Disease cycle of cherry leaf spot.

Infection will not take place until the leaves have developed mature stomates (breathing pores) in the underside of the leaf (Fig. 78). The leaves usually do not unfold sufficiently for infection to occur until petal fall. However, in years when leaf development is ahead of blossom bud development, infection may occur slightly earlier than petal fall. A wet period of only a few hours is required for infection when favorable temperatures exist. Ascospores mature gradually over an extended period, and the supply of ascospores is not exhausted from the apothecium until mid-summer.

Development of visible lesions occurs in 5 to 15

Fig. 75. Small, circular, purple spots due to cherry leaf spot on the upper surface of sour cherry leaves (top) and white spore masses (acervuli) on the lower surface of sour cherry leaves in the center of the spots (bottom).

Fig. 77. Apothecia of the leaf spot fungus on overwintered sweet cherry leaves.

days, depending on temperature and moisture conditions. Optimum conditions for lesion development are temperatures of 60 to 68°F with rainfall or high humidity. Soon after the lesions appear, the acervuli, containing flesh-colored masses of spores (conidia), are visible on the under surface of the leaf (Fig. 79). The conidia are readily carried by splashing rain and air currents to other leaves in the tree. Secondary spread and infection by conidia continues repeatedly during favorable weather until leaf fall in autumn.

Control

The disease cycle could be broken and the disease eliminated by completely preventing the fungus from overwintering on fallen leaves. Currently this is not feasible except for small backyard plantings.

The primary approach to the control of leaf spot is use of fungicide sprays. Applications are started at petal fall and repeated every 10 to 14 days to harvest. Often a postharvest application is made 2 to 3 wk after harvest.

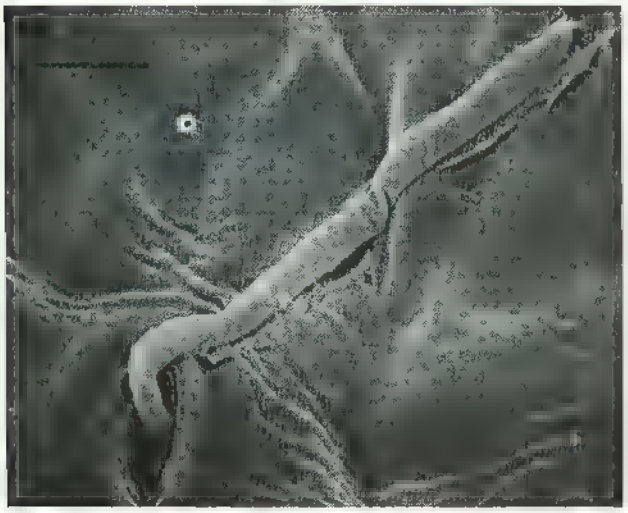

Fig. 78. Germinated conidium of the leaf spot fungus infecting through a stomate on the underside of a sour cherry leaf.

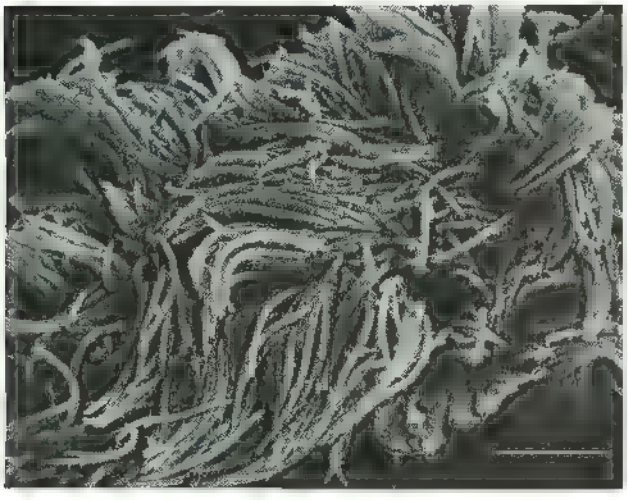

Fig. 79. Close-up of an acervulus containing the long, slightly curved conidia. The conidia are disseminated during rain to other leaves where they can infect.

HELPFUL REFERENCES

Eisensmith, S. P. and A. L. Jones. 1981. A model for detecting infection periods of *Coccomyces hiemalis* on sour cherry. Phytopathology 71:728-732.

Keitt, G. W., E. G. Blodgett, E. E. Wilson, and R. O. Magie. 1937. The epidemiology and control of cherry leaf spot. Wis. Agr. Exp. Sta. Res. Bull. 132. 118 pp.

Peach Scab

Peach scab occurs primarily in the warmer peach producing areas. It is worst where a good spray schedule is not followed early in the season. The causal fungus, *Cladosporium carpophilum*, also attacks apricot and nectarine.

Symptoms

Although the disease occurs on the twigs and leaves, it is most conspicuous on the fruit. Early infections appear as small, greenish, circular spots on the surface of the fruit. These spots do not generally appear until the fruit are half-grown and tend to be concentrated at the stem end. Older lesions become black and velvety in appearance as spores are pro-

Fig. 80. Peach scab lesions on the fruit.

duced (Fig. 80). On apricot, the lesions are more brown than black. Lesions may run together when numerous, resulting in abnormal growth of the fruit, and in severe cases, cracking of the skin and flesh.

Disease Cycle

The fungus overwinters in lesions on the twigs. At 100% relative humidity, 20 to 30 hr are required for abundant sporulation in lesions on twigs. Conidia are airborne and waterborne, and are most abundant 2 to 6 wk after the shuck split stage of development. If weather conditions are favorable, infection begins to occur at about shuckfall. The fruit remain susceptible until harvested. Forty to 70 days elapse from the time the spore lands on the fruit till the disease is visible. Thus, the disease is usually not observed until the fruit are well grown. Spores from the fruit reinfect the twigs and leaves.

Control

The disease is controlled primarily with fungicide sprays, although pruning helps increase air circulation and reduces the chances for infection. The first spray should be applied 10 to 12 days after the shuck split stage of bud development on peach, somewhat earlier on apricot and nectarine. Fungicide protection is especially critical 2 to 6 wk after shuck split. Sprays should be applied every 10 to 14 days until 40 days before harvest. Because of the long incubation period, infections up to 40 days before harvest will not appear before the fruit are marketed and consumed.

HELPFUL REFERENCES

Keith, G. W. 1917. Peach scab and its control. U.S. Dept. Agr. Bull. 395, 66 pp.

Lawrence, E. G., Jr. and E. I. Zehr. 1982 Environmental effects on the development and dissemination of *Cladosporium carpophilum* on peach. Phytopathology 72:773-776.

Bacterial Spot

Bacterial spot is a problem on susceptible apricot, peach, nectarine, prune and plum varieties. It causes severe defoliation and fruit spotting, which weakens the trees and makes the fruit unmarketable.

The disease was originally described by Erwin F. Smith in 1902 on Japanese plums from Michigan. He attributed it to a bacterium known today as *Xanthomonas campestris* pv. *pruni*. This disease is widespread in the eastern United States.

Symptoms

The bacteria infect the leaves, fruit and tender growing shoots. Leaf lesions are small and generally angular in outline. Initially, lesions appear as water-soaked areas primarily on the underside of leaves, later as brown to black spots. Often the centers of spots fall out, and margins have a reddish coloration. The disease is generally worst at the tip of the leaf, where an inch or more may be killed (Fig. 81).

Severely infected leaves soon turn yellow and fall to the ground. On sensitive varieties, a few lesions result in severe defoliation; tolerant varieties require many more. Heavy defoliation early in summer reduces fruit size and weakens the tree.

Fruit infected early in the growing season develop cracks or checks in the skin (Figs. 82-84). Lesions extend into the flesh, resulting in deep pits, which must be removed during processing. Under certain weather conditions, fruit lesions show gumming.

Fig. 81. Peach leaves with tip yellowing from infection by bacterial spot.

Fig. 82. Bacterial spot infection to Suncling peach fruit.

Fig. 83. Bacterial spot on apricot.

Fig. 84. Bacterial spot on Stanley prune fruit.

Late season infections are superficial, giving the fruit a mottled appearance.

Infection to the current season's growth can result in the production of two types of cankers. Lesions apparent in the year of infection are called summer cankers. These are usually located between the nodes. Infections occurring in the autumn, but not visible until the following spring, are called spring cankers. These tend to be found at buds or nodes.

Disease Cycle

Overwintering occurs in spring cankers. Recent evidence indicates these are initiated primarily through leaf scars. The scars left by late-maturing leaves are most susceptible to infection. Survival of bacteria in summer cankers of peach is rare in northern states, more common in the South. On plum and apricot, bacteria may survive in summer cankers for more than one season.

When canker development is resumed in spring, the bacteria ooze out of the lesions and are spread by windblown rain to younger leaves, fruits or shoots. Moisture from fog and dew is important in the dissemination process on individual leaves. Hard, driving rains are more important than gentle rains in initiating new infections. This explains why bacterial spot can be proportionally more severe on one side of the tree than on the other.

Secondary spread from summer cankers, leaf lesions and fruit infections continues whenever environmental conditions permit. Periods of frequent rainfall with moderate temperatures and fairly high winds favor infection. The disease may become quite severe if these conditions persist through June and July. Extended periods of hot, dry weather reduce disease spread and development.

Control

Use of resistant varieties is the primary method for controlling bacterial spot. Some of the more susceptible varieties are: Blake, Jerseyland, Suncrest, Suncling, Sunhigh and Jersey Queen. J. H. Hale, Babygold 5, Elberta, Kalhaven and Rio-Oso-Gem are moderately susceptible. Relatively resistant varieties: Redskin, Redhaven, Loring, Candor, Biscoe, Dixired, Sunhaven, Jefferson and Madison.

Most apricot varieties are susceptible, as well as many nectarine varieties.

Know a variety's relative susceptibility to bacterial spot before buying. Bacterial spot problems can also be reduced by locating new plantings away from older ones containing susceptible varieties. Planting susceptible peach, apricot, prune and nectarine orchards in close proximity to one another can contribute to buildup of this disease.

Balanced fertilization programs should be followed. Excess nitrogen may aggravate the problem, while trees weakened from poor nutrition or other causes are more sensitive to the disease

Some spray programs help control bacterial spot. However, these programs only suppress development of the disease they do not eliminate it. Because of the cost and uncertainty of chemical control, the best way to control bacterial spot is through the use of resistant varieties.

HELPFUL REFERENCES

Feliciano, A. and R. H. Daines. 1970. Factors influencing ingress of *Xanthomonas pruni* through peach leaf scars and subsequent development of spring cankers. Phytopathology 60:1720-1726.

Miles, W. G., R. H. Daines, and J. W. Rue. 1977. Presymptomatic egress of *Xanthomonas pruni* from infected peach leaves. Phytopathology 67:895-897.

Fig. 85. Leaf curl on peach leaves.

Peach Leaf Curl

Leaf curl is a springtime disease of peach and nectarine that can result in severe defoliation, weakened trees, and reduced fruit quality, set and yield.

Symptoms

The disease is most noticeable on the leaves within a month after full bloom. Infected leaves are thickened and puckered and often flushed with red or purple (Fig. 85). Thickening and puckering may be limited to small areas of the leaf or may involve the entire leaf. Severely infected leaves are two to three times larger than healthy leaves and exhibit extensive curling. A few to most leaves on a tree may be infected. Old infected leaves become necrotic, wither, and fall from the tree.

Infected shoots are stunted, swollen, chlorotic and rosetted. Symptoms are confined to the new growth. Infected flowers are distorted and usually drop before the symptoms are well developed.

Infected fruit exhibit raised, wrinkled areas with reddish discoloration. Infected fruit often fall prematurely.

Causal Agent

Soon after the distorted leaves are visible, asci of the leaf curl fungus *Taphrina deformans* break through the cuticle (Fig. 86). The asci appear as a powdery-gray felt-like area on the thickened leaf. Each ascus contains ovoid ascospores, which are released when the mature asci rupture. During periods of wet weather, the ascospores produce large numbers of blastoconidia or budconidia by budding.

Disease Cycle

Infection occurs during the spring from bud swell to bud opening. Spring rains wash ascospores from overwintering sites on the surface of the host to the surface of leaf buds. The spores continue to multiply during periods of moist weather. Once the bud scales loosen, the small bud conidia are carried in films of water to the leaf tissue where infection takes place.

Environment can limit leaf curl infection—an explanation of why the disease does not occur every year. Leaf curl is worse when the weather is cool and wet. Low temperatures are thought to retard maturation of leaf tissue, thus prolonging the time infection may occur. The fungus can penetrate young peach leaves readily at temperatures between 50 and 70°F, but only weakly below 45°F. Rain is necessary for infection.

Control

A single spray, using the correct material, if applied before bud swell, will provide nearly perfect control of leaf curl. Autumn sprays after the majority of the leaves have fallen or spring sprays within 3 to 4 wk of bud swell are effective, if applied thoroughly. Once the fungus enters the leaf, the disease cannot be controlled.

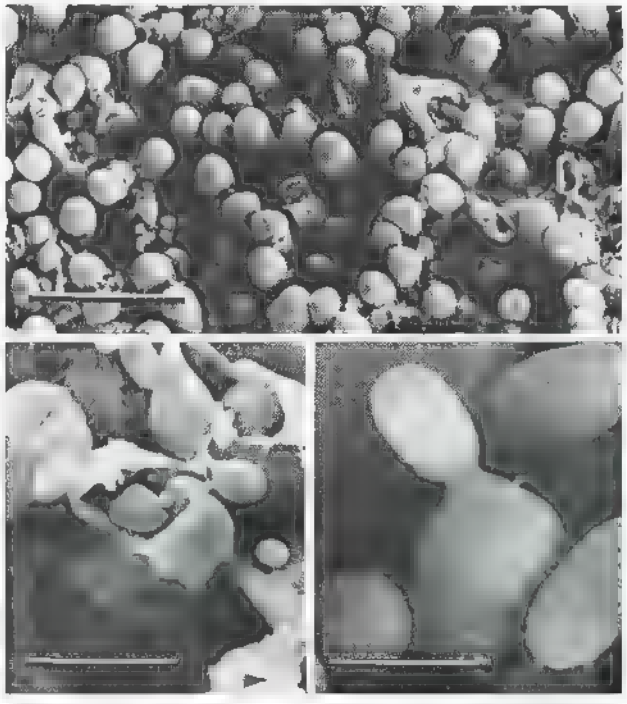

Fig. 86. Asci of the leaf curl fungus emerging from the surface of a leaf (top). Ruptured ascus containing ascospores and producing a bud conidium (bottom, left). An ascospore producing a bud conidium (bottom, right).

No variety is immune to leaf curl, but Redhaven and most varieties derived from Redhaven have some tolerance to leaf curl. Redskin and varieties derived from Redskin are more susceptible than Redhaven. To help maintain vigor where leaf curl is severe:

1) thin fruit heavily to reduce the demand on the remaining leaves;

2) reduce drought stress by periodic irrigation, and

3) apply a nitrogen fertilizer. (The amount of fertilizer should be controlled so that the trees are not overstimulated.)

HELPFUL REFERENCES

Fitzpatrick, R. E. 1934. The life history and parasitism of *Taphrina deformans*. Sci. Agr. 14.305-326

Mix, A. J. 1935. The life history of *Taphrina deformans*. Phytopathology 25.41-66.

Ritchie, D. F. and D. J. Werner. 1981. Susceptibility and inheritance of susceptibility to peach leaf curl in peach and nectarine cultivars. Plant Dis. 65-731-734

Plum Pockets

This disease, caused by *Taphrina pruni*, is similar to peach leaf curl, but attacks plum. Plum pockets occurs to some extent on wild or unsprayed trees, rarely in commercial plantings. It is recognized by the production of abnormally large, misshapened, bladder-like fruit with thick spongy flesh (Fig. 87). The centers of infected fruit are hollow due to the failure of the young seed to develop. The surface of the fruit is covered with a powdery-gray fungal growth due to the projection of asci through the cuticle or waxy layer covering the flesh of the fruit. Old infected fruit become brown, wither, and fall from the tree.

Because this disease is not of commercial importance, chemical controls have not been developed. A spray program similar to the one for peach leaf curl may control plum pockets as well.

Fig. 87. Plum pockets. Note enlarged, infected fruit on left and normal-size healthy fruit on right.

Powdery Mildew of Cherry

Mildew, caused by *Podosphaera oxyacanthae*, is a serious problem on cherries in the nursery, an occasional problem in sour cherry orchards and a rare problem in sweet cherry orchards. Mildew can reduce the growth of young trees and cause defoliation of older trees.

Symptoms and Disease Cycle

Mildew on young leaves appears as white, felt-like patches with a tangle of mycelium and spores. Lesions spread rapidly, engulfing the entire leaf (Fig. 88). Severely infected leaves are folded upwards, become stiff and brittle with age and often drop to the ground prematurely. Flower and fruit infections are rare, although young fruit are reported susceptible in areas of the western United States.

Conidia are produced in chains and are carried by wind to the new growth where they infect. The surface growth is supported by haustoria in the epidermal cells of the leaf. Beginning at mid-season, numerous small, brown to black, spherical specks (cleistothecia) develop in the felt-like patches (Fig. 89). The cleistothecia have several dichotomously branched appendages which are recurved at the tip. Each cleistothecium contains a single ascus with eight ascospores.

The fungus probably overwinters as mycelium in dormant buds and as cleistothecia in fallen leaves.

Control

Where powdery mildew is a problem on cherries, it is controlled with mildewcides applied during the summer months.

Black Knot of Plum

Black knot, caused by the fungus *Dibotryon morbosum*, is a serious disease of plums and prunes. It is also commonly found on wild plum and cherry trees, which serve as reservoirs of inoculum for nearby commercial orchards. Losses result from dieback of girdled limbs and stunting of the growth above the knots.

Symptoms

The disease is characterized by the production of elongated swellings or knots on the limbs of susceptible prune and plum (Fig. 90). These corky outgrowths predominate on small twigs and branches but may

Fig. 88. Powdery mildew-infected shoot of sour cherry. Note white growth on leaves and distortion.

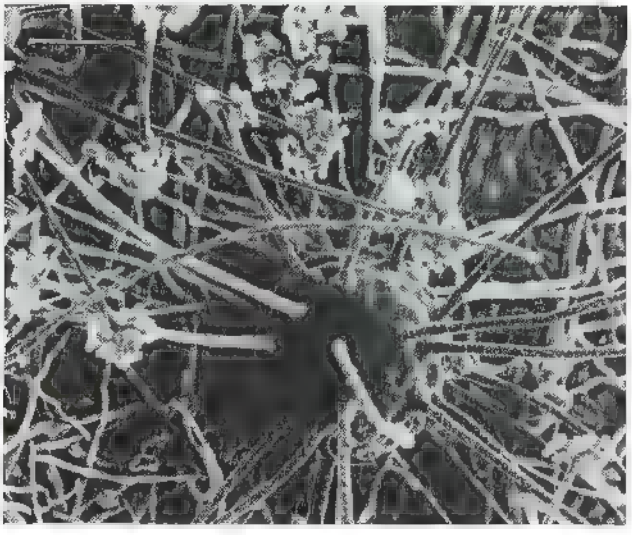

Fig. 89. Cleistothecium of cherry mildew with dichotomous branching of appendages.

Fig. 90. Severe black knot infection to scaffold branches of prune.

Fig. 91. Black knot on branches of prune.

also be located on larger scaffold branches and on the trunk. Knots, which tend to be longer than wide, may reach a foot or more in length.

Newly formed knots are greenish and soft but become hard and black with age (Fig. 91). Old knots may be covered with a white or pink parasitic fungus during summer and be riddled with insect borers.

Disease Cycle

In spring, at the green tip stage of bud development, ascospore discharge is initiated from perithecia located along the surface of the knots. Spore discharge is moderate to heavy from pink to 2 wk after bloom, and ends about the time terminal growth stops. Rain is required to discharge the spores. Spores are carried by wind and rain to the site of infection. Infection takes place through the unwounded surface and is most severe when wet conditions are accompanied by temperatures between 55 and 77°F. Infection continues to occur until terminal growth stops or the supply of spores is exhausted.

Several months are required between initiation of infection and appearance of knots. Some knots are visible by late summer, others not until spring the next year. At least 1 yr (usually 2) is required before new knots produce mature perithecia. The fungus in old knots is active during fall and early spring months, extending the knots several inches in length. This advancement is usually not visible until late spring.

Varietal Susceptibility

The two major plum varieties grown east of the Rocky Mountains, Stanley and Damson, are susceptible to black knot. Bluefre is also susceptible. Varietal susceptibility tests in Pennsylvania show Shropshire and Stanley are highly susceptible; Methley, Milton,

Early Italian, Brodshaw and Fellenburg moderately susceptible; Shiro, Santa Rosa and Formosa slightly susceptible and President apparently resistant to the black knot fungus.

Control

Plans for preventing the disease from building up in new plantings should be developed before the orchard is established. Wild plums and wild seedling cherries, other than black or pin cherry, should be removed from fence rows and nearby wooded areas. Establish and maintain at least a 600-ft border free of wild hosts. Do not plant new plum orchards next to or downwind from old plantings with black knot. These simple precautions will greatly reduce problems with the disease and increase orchard longevity.

Once established, the orchard and surrounding wooded areas should be inspected annually for infection. Although infections are more difficult to find in mid-August, removal of knots at this time is desirable because they are generally fully extended.

Make cuts 2 to 3 inches below the swelling. Remove knots on the trunk or main branches by cutting away the diseased tissue down to the wood and at least 1/2 inch outward beyond the margin. When infection is severe, leave knot removal until late winter or spring so as not to increase vigor excessively. Since the fungus may have extended beyond the swelling, make cuts well below the visible infection. Remove the knots from the orchard and burn before April 1 since they can serve as a source of inoculum if left on the orchard floor.

Spraying, if done in conjunction with a sanitation program, will help prevent the disease. Spraying alone will not control the disease.

HELPFUL REFERENCES

Smith, D. H., F. H. Lewis, and S. H. Wainwright. 1970. Epidemiology of the black knot disease of plums. Phytopathology 60:1441-1444.

Wainwright, S. H. and F. H. Lewis. 1970. Developmental morphology of the black knot pathogen on plum. Phytopathology 60:1238-1244.

Fig. 92. Powdery mildew lesions on surface of young peach fruit.

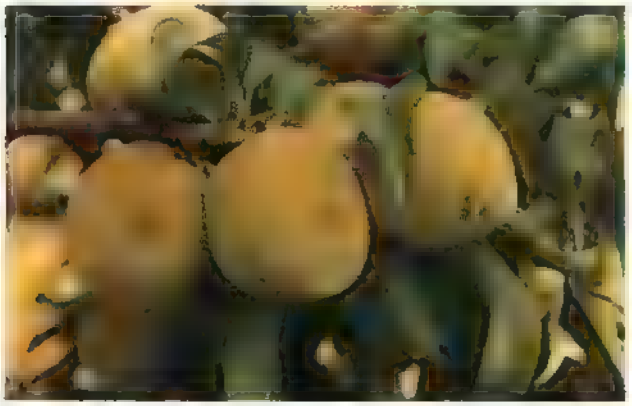

Fig. 93. Rusty spot of peaches probably caused by powdery mildew.

Rusty Spot/Powdery Mildew of Peach

Powdery mildew is rarely a major problem in midwestern and northeastern peach orchards. It may lead to some culling of fruit in years when weather conditions are favorable for infection. The problem is greatest on susceptible varieties and in orchards where sulfur has been dropped from the spray program.

The causal organism is generally considered to be the fungus *Sphaerotheca pannosa*. However, *Podosphaera oxyacanthae*, the common mildew species on cherries, also attacks peach. Due to difficulty in separating the species with certainty, the relative importance of each is not known.

Symptoms

In the orchard, the disease is observed primarily on the fruit. Lesions on young fruit appear as white powdery spots (Fig. 92). As the fruit mature and the fungus growth responsible for the early coloration disappears, the spots look hard and leathery. The dark brown or "rusty" spot is often observed on the mature fruit at harvest (Fig. 93).

Leaf and shoot symptoms may occur on nursery stock or on new shoots of older trees. Infected leaves are coated with a thick layer of fungus mycelium and become distorted and narrow. Shoot growth is stunted. Foliar symptoms are generally less prevalent in peach mildew than in apple and cherry mildew.

Disease Cycle

The mildew fungus overwinters as mycelium in dormant peach buds. When infected shoots emerge in spring, the pathogen initiates its growth and produces spores (conidia). The spores are carried to the leaves, twigs and fruit by wind and rain. Infection may also be initiated in the orchard by spores produced outside the planting. Other stone fruit crops and roses are common external sources of disease.

Fruit have been observed to be most susceptible when young, but they become resistant with pit hardening. Lesion extension continues for a short time after resistance to infection develops. New leaves can be infected throughout most of the season but become resistant with age. The disease is most severe following periods of moderate temperatures and high humidity. Spore germination will occur at relative humidities of 43 to 100%. Free moisture is not required. Optimum temperature for germination is 66 to 72°F. Under favorable environmental conditions, new lesions develop in 10 days.

Control

Problems with this disease can be eliminated by avoiding mildew-susceptible varieties. Reo-Oso-Gem and Redskin are susceptible. If these varieties are planted, supplemental sprays with mildewcides are necessary for control.

Fungicide sprays should be initiated at petal fall and reapplied periodically until the pit hardening stage of fruit development. If a severe problem exists, chemicals can also be used to reduce the severity of leaf and shoot infection. For this purpose, start spray applications early and continue until growth stops.

HELPFUL REFERENCES

Ries, S. M. and D. J. Royse. 1978. Peach rusty spot epidemiology: incidence as affected by distance from a powdery mildew-infected apple orchard. Phytopathology 68:896-899

Weinhold, A. R. 1961. The orchard development of peach powdery mildew. Phytopathology 51:478-481.

Fusicoccum Canker of Peach

Fusicoccum canker, caused by *Fusicoccum amygdali*, is a minor disease of peach that occurs primarily in peach growing areas of the Atlantic Coastal region from Massachusetts south. In early summer, infected twigs begin to wilt and die, and sunken cankers are found at the base of the current season's growth (Fig. 94). Gumming is commonly

Fig. 94. Death of peach shoots due to Fusicoccum canker (top) and close-up of cankers at the base of the shoots (bottom).

associated with cankers, but it is not a good diagnostic characteristic because other canker pathogens also cause gumming. Darkly pigmented pycnidia are produced over the surface of the cankered area and contain single-celled, fusoid and hyaline conidia. Under moist conditions, the conidia ooze from the pycnidia. Fusicoccum may also produce a leaf spot and infect the fruit.

HELPFUL REFERENCES

Guba, E. F. 1953. Large leaf spot and canker of peach caused by the fungus *Fusicoccum amygdali* Delacr. Plant Dis. Rep. 37:560-564.

Gummosis

Gummosis was first reported on peaches near Fort Valley, Georgia, in the late 1960's and early 1970's. It is caused by three species of *Botryosphaeria: B. dothidea, B. obtusa (=Physalospora obtusa)* and *B. rhodina*. Today the disease is reported throughout central Georgia, central Alabama, Florida and Louisiana.

Symptoms and Disease Cycle

Gummosis is characterized by numerous sunken necrotic lesions 1/4 to 1/2 inch in diameter around lenticles and by excessive gum exudation (Fig. 95). Removal of the bark reveals shallow, round to oval, brown, gummy lesions 1/2 to 1 inch in diameter. On young branches lenticles become swollen, but gumming does not occur. Symptoms initially occur on the trunk between the ground and the scaffold limbs usually during the second or third growing season. The disease later affects the scaffold limbs and twigs. Severe infection may kill twigs, reducing fruiting wood. Ascospores and conidia are produced in stroma in infected lenticles.

The *Botryosphaeria* spp. which cause this disease have many hosts. Conidia and ascospores produced in fruiting bodies in cankers on peach trees or other hosts are released during rain and are washed or blown into the peach branches and infect through lenticles. Infection is greatest during the summer months. For identification of *B. dothidea* and *B. obtusa* see apple white rot and black rot, respectively.

Fig. 95. Extensive gum exudation on the trunk of a peach tree (gummosis) caused by *Botryosphaeria*.

Control

Destruction and removal of dead trees and prunings may be helpful in reducing inoculum. There is no chemical control for the disease.

HELPFUL REFERENCES

Weaver, D. J. 1974. A gummosis disease of peach trees caused by *Botryosphaeria dothidea*. Phytopathology 64:1429-1432.

Bacterial Canker

Two closely related bacteria, *Pseudomonas syringae* pv. *syringae* and *P. syringae* pv. *morsprunorum*, cause this disease. Bacterial canker has been found primarily on sweet cherry but can be serious on sour cherry, peach and plum.

Symptoms

The disease attacks most parts of the tree. Branches and twigs are cankered, and a heavy exudation of gum is usually associated with these cankers in the autumn and again in the spring (Fig. 96). Leaves on the terminal portions of severely cankered branches may wilt and die in summer or early autumn from girdling. Under severe conditions, large scaffold limbs may be killed.

Leaf and fruit infection are relatively common

Fig. 96. Gum exudation on a young tree infected with bacterial canker.

under certain conditions. They appear to be most severe in prolonged periods of wet, cool weather after bloom. Infected leaves develop dead areas or spots which drop out, giving them a tattered appearance (Figs. 97, 98). Severely infected leaves fall to the ground prematurely. Deep, black depressions develop in the flesh of young cherry fruit and on the stems of the fruit (Fig. 99). Infected fruit are more susceptible to brown rot than healthy fruit and may contribute to outbreaks of the disease.

Leaf and flower buds may be killed while dormant, thus failing to open in the spring. Small cankers sometimes develop at the base of diseased buds. This symptom has also been observed on peach. In some cases, infected spurs open normally, but collapse in early summer, leaving wilted leaves and dried up fruit. Blighting of spurs year after year eliminates bearing surface and makes trees worthless.

Fig. 97. Severe leaf rolling and spotting along the leaf margin of sweet cherry leaves from bacterial canker.

Fig. 98. Necrotic spots and yellowing of sour cherry leaves due to bacterial canker.

Fig. 99. Rot of immature sweet cherry fruit caused by bacterial canker.

Disease Cycle

The bacteria overwinter in cankers, infected buds and systemically in apparently healthy tissues of the tree. Infection occurs in the autumn through leaf scars and in spring through scars left by bud scales.

In spring, bacteria from cankers and infected buds are disseminated by rainfall onto emerging tissues. Infection to developing blossoms, leaves and fruit occurs through natural openings and wounds. Leaves and blossoms are probably more susceptible when injured by frost.

Secondary spread from primary infections occurs throughout the growing season and is important to the buildup of the disease. Periods of frequent rainfall, cool temperatures and high winds are most favorable for early season infection and spread. Later in the season, leaves are less susceptible and temperatures too high for bacterial activity. Disease development remains low until autumn when environmental conditions again become favorable for the disease.

Control

Extensive field observations indicate that the disease is troublesome on some varieties but not others. Hardy Giant, Schmidt and Windsor are susceptible and often severely damaged. Hardy Giant appears to be most susceptible and should not be planted. On other varieties, periodic removal of cankers helps to eliminate the disease. Copper compounds can be helpful for control.

HELPFUL REFERENCES

Jones, A. L. 1971. Bacterial canker of sweet cherry in Michigan. Plant Dis. Rep. 55:961-965.

Latorre, B. A. and A. L. Jones. 1979. *Pseudomonas morsprunorum*, the cause of bacterial canker of sour cherry in Michigan and its epiphytic association with *P. syringae*. Phytopathology 69:335-339.

Silver Leaf

This disease, caused by the fungus *Stereum purpureum*, attacks most tree-fruit crops but is usually of minor importance. It is most common on sour cherry, sometimes a problem on apple.

Symptoms and Disease Cycle

Affected branches on trees exhibit leaves with a dull metallic luster. These "silvered leaves" are due to a separation of the epidermis from the palisade layer of the leaf. Because of this separation, the upper epidermis of affected leaves peels back easily. If the disease is severe, affected leaves may have necrotic areas in addition to silvering.

The fungus causing this disease is located in the heartwood of the trunk and branches some distance from affected leaves. Infected heartwood is darkly discolored, and the fungus can be isolated from this discolored wood. Diseased trees become stunted, gradually lose vigor, but may stay alive for several years before dying. Evidence suggests the fungus produces a toxin that induces the silvery condition when translocated to the leaves.

Fungal fruiting bodies of *S. purpureum* develop on the bark of dead branches and dead trees and are useful in making positive diagnosis.

Control

Keeping trees in good vigor will help them withstand the effects of infection. Prune out and remove all dead limbs from the orchard. Avoiding large pruning cuts and making cuts close to the main axis help to prevent entry of the fungus into the tree.

Fig. 100. Typical peach canker. Note roll of callus growth.

Perennial Canker

Perennial (Cytospora/Valsa) canker is caused by two related fungi, *Cytospora cincta* and *C. leucostoma*. Both species attack peach, apricot, prune, plum and sweet cherry. The disease is common in peach orchards and is a frequent cause of limb dying and death of peach trees. It is less important on other stone fruit crops.

Symptoms and Disease Cycle

Perennial cankers are oval to linear in outline and eventually are surrounded by a roll of callus at the margins (Fig. 100). Cankers enlarge gradually on a year-to-year basis until the limb or trunk is completely girdled. Active cankers often have gum associated with them, but "gummosis" is not unique to canker, since it may be caused by several unrelated factors.

The fungi overwinter in cankers or on dead wood. Pycnidia are located in diseased tissue under the bark. Conidia are produced in tendrils from the pycnidia and are disseminated by splashing and wind-driven rain. Infection is through damaged or injured bark. Cold injury is the most important factor predisposing trees to canker (Fig. 101), but pruning wounds, mechanical damage, insect punctures and leaf scars are other entry points (Fig. 102).

Moisture is required for spore germination. The rate of canker development following infection depends on temperature and the species of fungus involved. *Cytospora cincta* is favored by lower temperature than *C. leucostoma*. For example, optimum temperatures for growth of *C. cincta* and *C. leucostoma* are approximately 68 and 86°F, respectively. When temperatures are not favorable for fungal activity, callus formation occurs. Canker activity resumes when temperatures again favor the fungus.

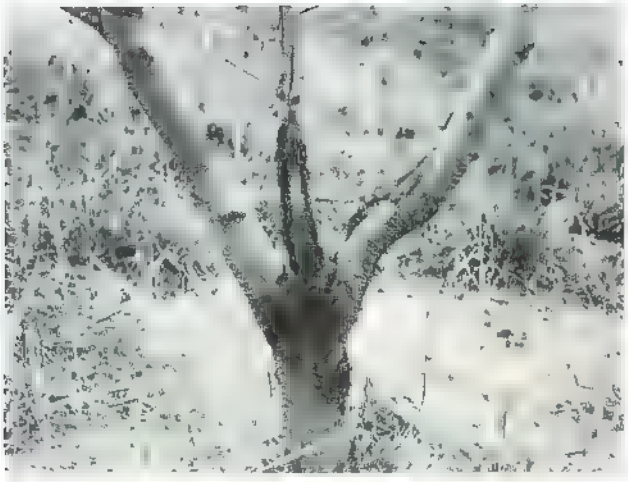

Fig. 101. Peach canker in crotch angle of tree following low temperature damage.

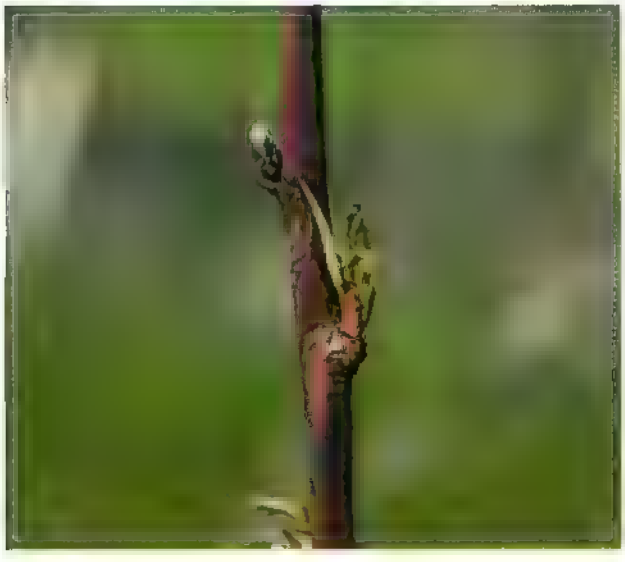

Fig. 102. Cankers initiated around the node of peach.

Control

Because of the manner of infection and development of this disease, no single control measure is adequate. Most known control methods act indirectly by reducing points of entry or by reducing the level of inoculum. Control can be facilitated by following these guidelines:

- —Avoid soils with poor internal drainage and remove wet spots by tiling before establishing new plantings.
- —Do not plant young peach orchards or replant trees next to older orchards or trees with cankers.
- —Delay orchard pruning until growth starts in spring. Moderate to severe pruning in November, or earlier, can severely weaken or kill trees. Late pruning promotes quick healing.
- —Eradicate cankers and remove badly cankered limbs, branches or trees. Burn cankered limbs soon after pruning. Sanitation is a must during the early life of the orchard.
- —In cultivated orchards, plant a cover crop by July 1 and mow thereafter as needed.
- —Fertilize early according to local recommendations.
- —Try to avoid mechanical and insect injury and do not leave long pruning stubs.
- —Apply fungicide sprays after pruning, but before rain.
- —Avoid weak-angled crotches when shaping trees.
- —Apply white latex paint to the southwest side of trunks and lower scaffold branches to help avoid cold injury.

HELPFUL REFERENCES

Hildebrand, E. G. 1947. Perennial canker and the canker complex in New York, with methods of control. Cornell Univ. Agr. Exp. Sta. Mem. 276. 61 pp.

Tekauz, A. and Z. A. Patrick. 1974. The role of twig infections on the incidence of perennial canker of peach. Phytopathology 64:683-688.

Anthracnose

Anthracnose, caused by the fungus *Glomerella cingulata*, is also known by the common name of ripe rot. In most years it is a minor disease of peach in the southeastern United States.

Symptoms and Disease Cycle

Anthracnose begins as small brown spots 1/16 to 1/8 inch in diameter which slowly enlarge to 1 inch or more in diameter (Fig. 103). Older lesions are circular, brown, firm, slightly sunken and are often covered with concentric rings of salmon-colored spore masses. The disease is most severe on ripening fruit. Early symptoms of anthracnose may be confused with brown rot; however, brown rot lesions enlarge more rapidly than those of anthracnose.

Anthracnose is sporadic and often occurs only in scattered trees. Alternate hosts are thought to be important sources of inoculum. The disease has been severe where blue lupine was grown in the vicinity of peaches.

The disease is identified by the concentric circles of salmon-colored conidia produced in acervuli on lesions on infected fruit. For a more complete description of the pathogen see apple bitter rot.

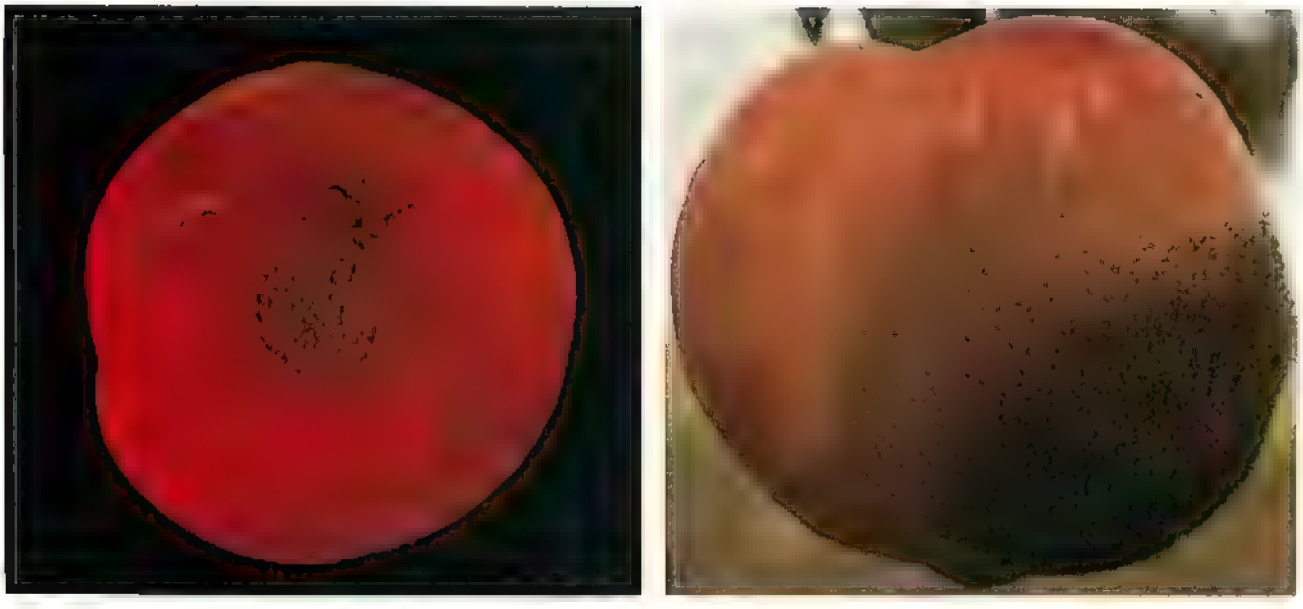

Fig. 103. Anthracnose (left) and Rhizopus rot (right) of peach.

Control

The disease is controlled by a fungicide used in cover sprays. Do not allow fruit to become overripe on the trees.

HELPFUL REFERENCES

Ramsey, G. B., M. A. Smith, and B. C. Heiberg. 1951. Anthracnose of peaches. Phytopathology 41:447-455.

Rhizopus Rot

This disease is important primarily when fresh peach, sweet cherry or plum are stored or otherwise delayed in reaching the consumer. The disease may develop in storage, transit or market place, depending on temperature and moisture conditions.

Symptoms and Disease Cycle

A rot very similar to brown rot develops on the surface of fruit. The skin of fruit affected with Rhizopus rot readily slips from the flesh underneath, while skin on a brown rot lesion does not. If humidity is low, infected fruit turn black and shrivel into mummies. Under more humid conditions, the infected fruit release liquid and become covered with a "whisker-like" growth (Fig. 103).

Infection may occur in the orchard at harvest time or in post-harvest handling of fruit. Injured fruit are most susceptible. However, once the disease becomes established, it can move onto sound fruit nearby. The fungus invades the tissue quickly, producing tufts of whisker-like fungal growth over the lesion. Black spherical structures (sporangia), each containing thousands of small sporangiospores, are produced at the tip of the bushy growth. The outer wall of the sporangia ruptures easily, freeing the sporangiospores, which then float about in the air or drop to the surface.

When environmental conditions are not favorable, dark colored spores (zygospores) are formed. These spores have a thick cell wall and can survive periods of dryness or unfavorable temperatures.

Control

Many of the practices used to control brown rot aid also in the control of Rhizopus rot. Careful handling of the fruit to avoid wounds, clean storage containers and warehouse facilities, and proper hydrocooling reduce the incidence of the disease. Fungicide sprays applied near harvest, or post-harvest dips, sprays or impregnated wraps are effective control procedures.

Alternaria Fruit Rot

Alternaria rot, caused by *Alternaria* spp., occurs on sweet and sour cherries. In the orchard, it is a minor problem except where fruit become overripe before harvesting or where cracking or other kinds of injury expose the flesh of the fruit to infection. Le-

sions are circular to oblong and slightly sunken; later becoming firm, flattened and wrinkled; and often dark-green to black because of abundant sporulation by the pathogen (Fig. 104). Lesions cover 1/3 to 1/2 of the fruit. The conidia are dark with both cross and longitudinal septa and are typically produced in chains.

In cold storage, rot often starts in the cavity left when the stem is detached from the fruit, in cracks of the skin and flesh, or in bruised areas. The disease is worst on overripe or fruit harvested late in the season.

Under cold storage conditions, the fungus produces fluffy, gray-to-white strands of mold over the surface of the lesions. No effective control measures are available except to harvest the fruit before it becomes injured or overripe.

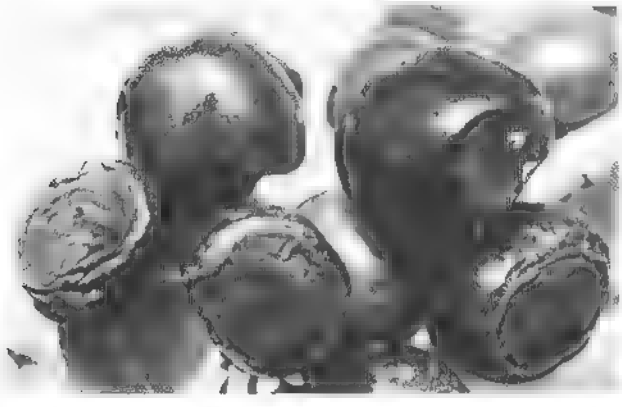

Fig. 104. Alternaria rot of sweet cherry fruit. Note flattened and wrinkled nature of the lesions.

Peach Tree Short Life

Peach tree short life is the most serious disease affecting peaches in the southeastern United States. It is a disease complex involving several pathogens, environmental factors and cultural practices. The disease is characterized by the sudden death of trees above the soil line in the late winter or early spring. These symptoms separate it from other factors which can affect peach tree survival, including root rots, wood decay fungi, drowning, etc.

Symptoms

Affected trees collapse suddenly after bloom or during early growth and are killed to the soil line (Fig. 105). In some instances trees may fail to bloom or leaf out, or symptoms may be delayed until early summer. Affected trees usually appear healthy the previous fall. Droplets of yellow or orange ooze may

Fig. 105. View of peach orchard where many trees have died due to peach tree short life.

be visible on the bark of the trunk or main scaffold limbs. The inner bark and cambium are discolored and a "sour sap" odor often characterizes these tissues.

Cause and Contributing Factors

Peach tree short life is caused by freeze injury or *Pseudomonas syringae* pv. *syringae* (the cause of bacterial canker) or a combination of both; affected tissues are often colonized by *Cytopsora* spp. Rootstocks, time of pruning, soil fumigation, nematodes and characteristics of the orchard site are involved in disease incidence. Trees propagated on root-knot-resistant rootstocks (Shalil, Yunnan, S-37, Nemaguard) are more susceptible than those planted on Lovell or Halford. Trees pruned in the autumn are much more susceptible than those pruned in the late winter or early spring.

The disease is much less severe where preplant and post-plant fumigation are used. High populations of ring nematodes are associated with peach tree short life. The disease is usually more severe where trees are planted in old orchard sites.

Disease Identification

Peach tree short life is usually diagnosed by the characteristic symptoms associated with it: sudden death and collapse of the tree in the spring. Freeze injury is characterized by a brown discoloration of the cambial layer, and, in severe instances, cracking and separation of the bark from the wood

Pseudomonas syringae pv. *syringae* infection is difficult to distinguish from freeze injury; infected bark is red-brown and cankers have a definite margin. When injured tissues are invaded by *Cytospora* spp., fruiting structures (pycnidia) are usually visible just beneath the bark. Under moist conditions, conidia, produced in thread-like strands, extrude from the pycnidia.

Control

A 10-point program has been developed for control of peach tree short life. The three most important elements of the program are:
— Prune as late as possible,
— Plant trees propagated on Lovell or Halford rootstock, and
— Use preplant soil fumigation.

If a material is registered, post-plant soil fumigation is also important. Other elements of the program include liming for proper pH, subsoiling prior to planting, maintaining a good fertilization program, using herbicides or shallow cultivation, and removing all dead or dying trees promptly.

HELPFUL REFERENCES

Ritchie, D. F. and C. N. Clayton. 1981. Peach tree short life: A complex of interacting factors. Plant Dis. 65: 462-469.

Crown Gall

Crown gall, caused by the bacterium *Agrobacterium tumefaciens*, is also known by the common names "root tumors" or "plant cancer". The disease affects a wide range of herbaceous and woody plant species, including stone fruit and pome fruit. The greatest disease incidence usually occurs in nurseries.

Symptoms

Crown gall is characterized by the formation of tumors or galls on affected plant parts. Galls begin as small, smooth growths which enlarge to become dark, hard, woody tumors with gnarled irregular surfaces. Tumors range from 1/4 inch to more than 4 inches in diameter (Fig. 106). Galls are typically globular but may be elongated or otherwise irregular. Old galls may be invaded by secondary fungi and insects. Galls are usually located on the roots or crown but, under certain conditions, may develop on the aerial portions of plants. Several galls may occur on the same root or stem

Disease Cycle

The bacteria which cause crown gall are widely distributed in soil where susceptible crops have been grown. New soils may become contaminated by planting nursery stock dug from problem soils. Once in the soil, the bacteria can survive for several years.

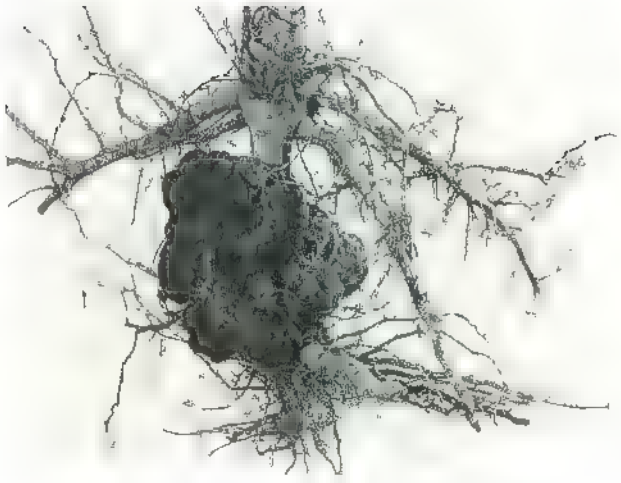

Fig. 106. Crown gall on Mazzard F 12/1 rootstock.

When susceptible crops are planted in infested areas, the bacteria enter the roots and crown through wounds produced in caring for, and handling, the nursery stock. They may also enter through wounds made by root feeding insects.

In addition to primary galls, secondary galls sometimes develop at some distance from the initial infection. These galls may develop on unwounded tissue, and bacteria cannot be found associated with them. Although the mechanism of secondary tumor formation is not entirely known, it is believed a tumor-inducing substance is produced in the primary gall and moves in the plant to induce secondary galls.

Pathogen Identification

Crown gall is identified primarily by characteristic galls formed on the roots and stems. Small galls could be confused with those caused by the root knot nematode. Root knot galls are usually smaller and associated with the fibrous roots. Several selective media are available for isolation purposes. Isolation is most successful from young galls.

Control

The use of disease-free nursery stock is extremely important to avoid the disease. Crown gall is severe on mazzard cherry and peach. It attacks other tree-fruit crops but is usually not as severe. Susceptible nursery stock should not be planted in soils with a history of crown gall. Crop rotation utilizing grain crops helps reduce the disease.

Biological control has been achieved with a non-pathogenic isolate of *A. radiobacter* var. *radiobacter*

(Strain 84). Root dips of nursery stock into a solution of *A. radiobacter* var. *radiobacter* have proved very successful in controlling the disease. Isolates of *A. tumefaciens* have been reported which are not controlled by this method. Soil fumigation does not successfully control the disease.

HELPFUL REFERENCES

Kerr, Allen. 1980. Biological control of crown gall through production of agrocin 84. Plant Dis. 64:25-30.

Schroth, M. N., A. R. Weinhold, A. H. McCain, D. C. Hildebrand, and N. Ross. 1971. Biology and control of *Agrobacterium tumefaciens*. Hilgardia 40:537-552.

Verticillium Wilt

Verticillium wilt, caused by the fungus *Verticillium dahliae*, can seriously affect cherry, peach and plum trees. Diseased trees are not often killed, but they persist as stunted and unproductive trees for several years.

Symptoms and Disease Cycle

The first symptom of infected trees is usually flagging of the leaves on one branch or perhaps the entire tree. Wilting usually begins in the middle of the summer, followed by browning and curling of the leaves and, ultimately, defoliation. Often true wilt does not occur, and the leaves simply turn yellow or brown and drop.

Symptoms usually develop on the lower part of the shoots first and progress upward, leaving a few green leaves at the tips of the shoots. The second year these shoots die back from the tip, partly as a result of winter injury.

In the sapwood of infected trees, brownish or grayish streaks occur (Fig. 107). This discoloration sometimes occurs in the current season's shoots, but usually is limited to the larger branches and the lower part of the tree.

Fig. 107. Grayish streaks in the sapwood of Montmorency sour cherry with Verticillium wilt *(courtesy K.G. Parker, Cornell Univ.).*

The fungus is a common soil inhabitant where it can survive for 2 to 3 yr in the absence of a susceptible host. It can build up to very high levels on susceptible weeds such as lambsquarter, pigweed, nightshade and ground cherry and on such crops as strawberries, raspberries, tomatoes, peppers and eggplants. The fungus invades through the roots of trees planted in infected soil.

Control

Since trees are not likely to become infected when the inoculum level in the soil is low, the most effective control measure is to prevent the buildup of the fungus in the soil. This involves cultivation of a nonsusceptible crop, such as a grass crop, on the site for 2 yr prior to setting a new orchard and maintaining good control of susceptible weeds. Preplant soil fumigation with a broad-spectrum fumigant that is active against fungi also helps reduce inoculum levels and gives the new trees a good start.

Adequate moisture and balanced nutrition to maintain good vigor will reduce the incidence of the disease and encourage the recovery of diseased trees.

HELPFUL REFERENCES

Parker, K. G. 1959. *Verticillium hadromycosis* of deciduous tree fruits. Plant Dis. Rep. Supplement 255: 39-61

Armillaria Root Rot

Armillaria root rot is caused by the mushroom-producing fungus *Armillaria mellea*. It is also known as oak root rot, mushroom root rot and shoe-string root rot. *Armillaria mellea* is widespread, has an extensive host range and affects all fruit crops. The disease occurs on lighter soils throughout the eastern fruit-growing areas.

Symptoms

Above-ground symptoms resemble those of other root rot diseases. Affected trees exhibit a premature reddening or purpling of the foliage in the fall, reduced terminal growth, and in more advanced stages, die back. One or two scaffold limbs may die initially, followed by death of the entire tree. Trees often die out in a circular pattern from a central point or localized area within the orchard.

White to slightly yellow mycelial growth of

A. mellea is evident within the bark of dead roots. A fan-shaped, fungal mat is often present between the bark and the wood. *Armillaria* is distinguished from other fungi by the presence of dark brown to black thread-like structures called rhizomorphs (Fig. 108). These structures are about the size of a "shoe-string" and often found on dead roots or on the surface of live roots in advance of the cankered area. On trees in more advanced stages of decline, clusters of mushrooms may arise at the base of the tree in autumn. These mushrooms are honey-colored with a ring or annulus on the stem or stipe just beneath the gills (Fig. 109).

Disease Cycle

Rhizomorphs and mycelial strands of fungus on infested roots enable the pathogen to survive many years in the soil. Rhizomorphs also function in spreading the fungus from tree to tree.

Control

Newly cleared forest land or fields with a history of Armillaria root rot should not be planted to susceptible crops. Cherry on mahaleb rootstock is the most susceptible fruit tree. Apple is moderately susceptible; pear is considered tolerant. Tolerant clones of Myrobalan, St. Julian and Methley plum are also known. If replanting infested tree sites is attempted, the soil should be well worked up and as many old roots as possible should be removed.

Fumigation has been only moderately effective in controlling the disease because the fungus can survive deep in the soil. This is particularly true where heavy soils restrict the movement of fumigants.

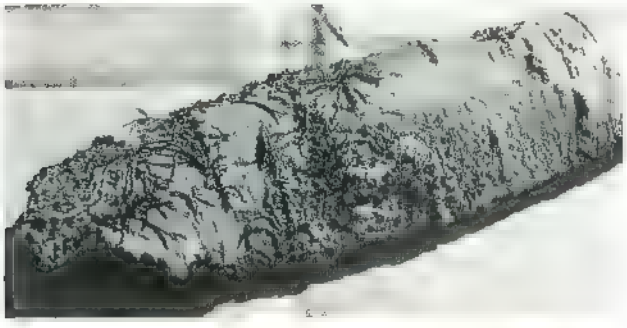

Fig. 108. Dark strands (rhizomorphs) of Armillaria on trunk of affected tree.

Fig. 109. Mushrooms at the soil line and white fungal mat beneath the bark are symptoms of Armillaria root rot.

HELPFUL REFERENCES

Wilbur, W., D. E Munnecke, and E. F. Darley. 1972. Seasonal development of Armillaria root rot of peach as influenced by fungal isolates. Phytopathology 62:567-670.

Zeller, S. M. 1926. Observations on the infections of apple and prune roots by *Armillaria mellea* Vahl. Phytopathology 16.479-484.

Clitocybe Root Rot

Clitocybe root rot, caused by *Clitocybe tabescens*, affects 210 species of plants, including apple, peach, cherry and plum. The disease is primarily important in the southern fruit growing regions of the United States.

Symptoms

Above-ground symptoms of Clitocybe root rot are similar to Armillaria root rot. Symptoms in infected roots are also similar except the white fungus mat which may develop beneath the bark of affected roots often has perforations in it (Fig. 110). Rhizomorphs are not formed. Rather, black, hardened mycelial extrusions often develop through cracks in the bark. In autumn during rainy periods, clusters of yellow to yellow-brown mushrooms may grow at the base of the affected trees (Fig. 111). These mushrooms are similar to those of Armillaria root rot but do not have an annulus around the stipe.

Pathogen Identification

Unless mushrooms or rhizomorphs are present, Clitocybe root rot is not easily separated from Armillaria root rot. The presence of perforations in the mycelial mat of Clitocybe is an aid in separating the two diseases.

Disease Cycle

Clitocybe tabescens can live for many years in dead roots in the soil. Live roots are infected when

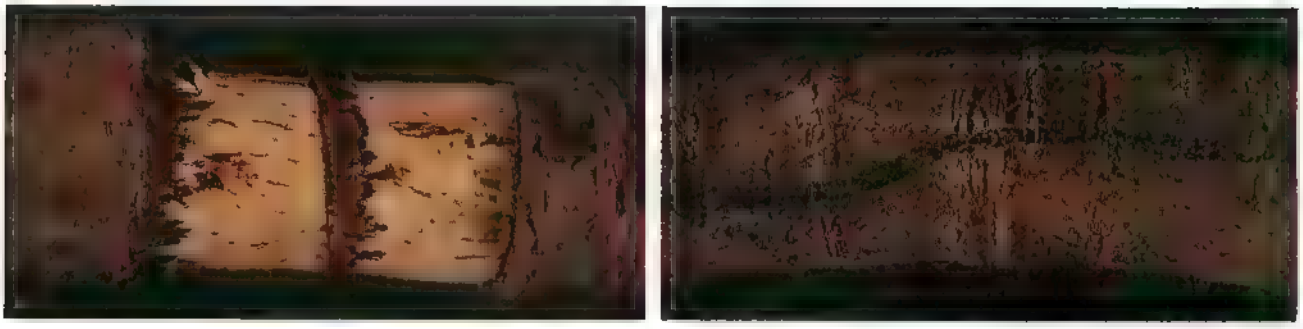

Fig. 110. *Clitocybe* **root rot. Perforations in the fungal mat on a root (left) help to separate** *Clitocybe* **from** *Armillaria*. **Hard, mycelial extrusions of** *Clitocybe* **develop through cracks in the bark (right).**

Fig. 111. Mushrooms of *Clitocybe*. **These are similar in color to those of** *Armillaria*, **but lack an annulus around the stipe.**

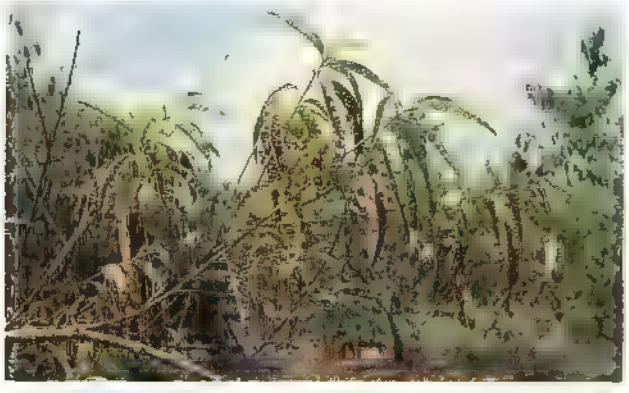

Fig. 112. Peach leaves with rolling, red blotches, and tattering due to X-disease. Note defoliation starting at the base of the shoot.

Fig. 113. Peach leaves yellowed and rolled from X-disease in late August.

they come in contact with the fungal extrusions in the dead roots.

Control

Control is the same as for Armillaria root rot.

HELPFUL REFERENCES

Rhoads, A. S. 1950. Clitocybe root rot of woody plants in the southeastern United States. U.S. Dept. Agr. Cir 853. Washington DC. 25 pp.

X-Disease

This disease occurs primarily in the Great Lakes states with distribution corresponding to the range of the wild chokecherry, a major reservoir of infection. It also occurs in the province of Ontario, Canada. Peach, nectarine, sweet cherry and sour cherry are affected by X-disease.

Symptoms

Peach: This disease is most easily identified on peach (Figs. 112-114). The first indication of its presence is a change in appearance of the foliage after about 2 mo of growth. Leaves on isolated branches curl inward and develop irregular yellow to reddish-purple spots. The spots soon drop out, leaving tattered leaves. Leaves on affected branches fall prematurely, starting at the base. Only a tuft of leaves remains at the tips of infected shoots.

Fruit-set may appear normal at first, but fruit on infected branches will drop prematurely. Symptoms

Fig. 114. Symptoms of X-disease randomly distributed throughout tree. Fruit on the affected parts drop soon after foliage symptoms appear.

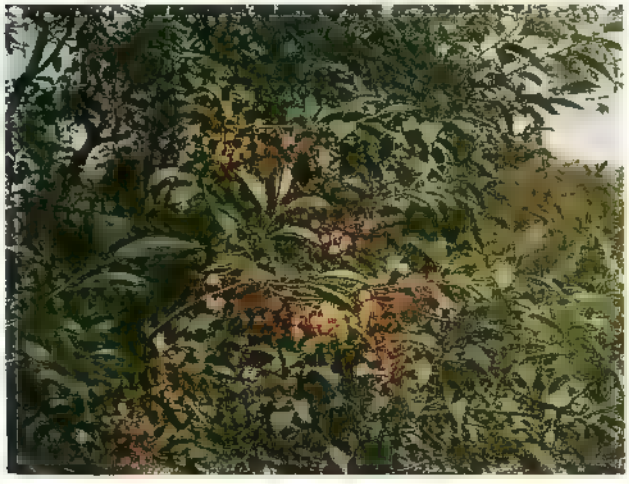

Fig. 115. Green to half-ripe cherries interspersed with normal fruit on a sour cherry tree on Mazzard rootstock infected with X-disease.

Fig. 116. *Paraphlepsius irroratus*, **one of several species of leafhoppers that can spread X-disease.**

will appear on isolated branches during the first and second years, but 2 to 3 yr after initial infection, most branches show symptoms.

Cherry: Cherries on mahaleb root are killed suddenly in midsummer by the disease. Trees on mazzard rootstock decline slowly. Scattered fruit on trees propagated on mazzard rootstock are small and pink at harvest time and have a bitter flavor (Fig. 115).

Cause

X-disease was believed to be caused by a virus until structures resembling mycoplasma were discovered in association with diseased plants. Mycoplasma are small parasitic organisms which have long been known to cause disease in animals and man. The organisms produce spherical-to-ellipsoid-shaped bodies that are smaller than bacteria but larger than most virus particles (Fig. 117).

Mycoplasma live in phloem cells of plants. The phloem is a network of cells for moving foodstuffs manufactured in the leaves to other parts of the plant. Mycoplasma are also sensitive to certain antibiotics, and some of these chemicals have shown promise for control.

Because the discovery of mycoplasma as plant disease agents is relatively recent, plant pathologists have taken a new look at several old peach diseases that were imperfectly understood. Besides X-disease, mycoplasma have been associated with peach yellows, little peach and peach rosette.

Spread of X-Disease

Extensive research has shown that X-disease can be transmitted by several species of leafhoppers (Fig. 116). These leafhoppers acquire the X-disease agent while sucking juices from the leaves of X-disease infected chokecherries. Two to three weeks later they can inject the agent into healthy leaves while feeding. These leafhoppers are usually not considered pests of peach and cherry. The main damage they cause is the incidental transmission of X-disease.

Spread by leafhoppers can also occur from infected sweet and sour cherry, particularly from trees on mazzard rootstock. Although the possibility of spread from peach to peach has been investigated many times, it appears to be of minor importance.

Control

Eradication of chokecherry near stone fruit orchards helps to control X-disease. Chokecherry bushes are commonly found in hedgerows, along pro-

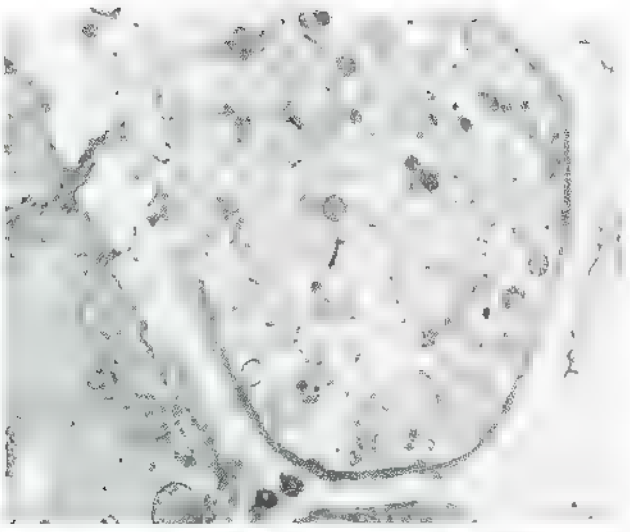

Fig. 117. Cross section of a vein in a peach leaf infected with X-disease. The spherical bodies are mycoplasma and are found in a phloem cell.

perty lines, in open woods and in overgrown meadows and abandoned fields.

Brush killers offer the cheapest and most effective way to kill the bushes with both summer and autumn spray treatments. Other removal methods include bulldozing, deep plowing, burning or pulling out individual bushes.

During the growing seasons following removal, check the treated area carefully for chokecherry sprouts. Sprouts or new chokecherry seedlings should be treated with herbicide sprays or pulled out during the summer.

Other controls include:

—Maintaining a vigorous insect control program from June through harvest with insecticides effective against leafhoppers.

—Removing infected peach and cherry trees in young orchards. Also remove infected cherry trees on mazzard rootstock. X-disease is often very severe in young peach orchards planted next to old cherry blocks with X-disease infected trees.

In older orchards (5-yr-old or above), injection treatments will provide disease remission. Contact your local extension agent for the antibiotics currently registered for control of X-disease.

Identification of Chokecherries

Chokecherry is often confused with wild black cherry and wild pin or fire cherry. It is important to be able to distinguish this species from others because only chokecherry is important in the spread of X-disease.

Unlike black cherry and pin cherry, which grow like trees to 50 ft or more, chokecherries are shrubs up to 15 ft tall. They are usually found in clumps. Their fruit, which is produced along a central stem, is black when mature and ripens before those of black cherry. The calyx cup does not persist as it does on black cherry (Fig. 118). Pin cherry fruit are borne in clusters like sour cherry and in no way resemble those of chokecherry. Chokecherry leaves are wider and broader than black cherry or pin cherry leaves. Serrations along the margin of the leaves are more prominent and spreading than those of the other two species.

HELPFUL REFERENCES

Gilmer, R. M., D. H. Palmiter, G. A. Schaffers, and F. L. McEwen. 1966. Insect transmission of X-disease virus of stone fruits in New York. NY State Agr. Exp. Sta. (Geneva) Bull. 813. 22 pp

Rosenberger, D. A. and A. L. Jones. 1978. Leafhopper vectors of peach X-disease and seasonal transmission from chokecherry. Phytopathology 68.782-790.

Sinha, R. C. and L. N. Chiykowski. 1980. Transmission and morphological features of mycoplasma-like bodies associated with peach X-disease. Can. J. Plant Pathology. 2.119-124

Stem Pitting of Stone Fruits

Prunus stem pitting was first noted as a severe problem on peach in areas of Maryland, Pennsylvania, and West Virginia in 1967. In the Great Lakes states, the disease exists in several scattered locations but has been less severe than in the mid-Atlantic states. In some locations, the disease has been traced to infected nursery stock, while in other locations, infection appeared to follow planting, indicating the disease agent was probably already present in the soil.

Fig. 118. Cluster of pin cherry fruit (left), chokecherry fruit (center) and black cherry fruit (right). Note that black cherry has a calyx cup which persists *(courtesy K.G. Parker, Cornell Univ.).*

Although the major problem has been with peach, apricot and nectarine, stem pitting affects other Prunus spp. as well. These include: European plum, Japanese plum, myrobalan plum, Chinese wild peach, Duke cherry, *P. mahaleb* and sour cherry.

Prunus stem pitting is caused by strains of tomato ringspot virus and is transmitted in the soil by the dagger nematode, *Xiphinema americanum*. It is also transmitted through infected nursery stock.

Symptoms

Foliar symptoms develop in late summer after harvest, but before the time of normal defoliation. Leaves on affected trees cup upward, become yellowish, later turn reddish-to-purplish and fall to the ground prematurely. Trees affected for a year or so develop leaf symptoms on one or two branches only. Later the entire tree shows symptoms.

Severely affected trees are stunted and weak. Their root system is poorly developed, and when trees are pulled out of the ground, socket-like depressions remain where roots have broken away. These trees may break off slightly below ground level from high winds, exposing the disorganized wood in cross section.

When the bark is removed from the wood below ground level, elongated pits or indentations are evident (Figs. 119, 120). Severity of pitting depends somewhat on the variety or clone of the rootstock and the stage of disease development. Closer examination of the crown area shows that the bark is 1 to 2 inches thicker than normal and has a spongy texture.

Control

To avoid introducing this disease into new plantings, purchase certified virus-free trees grown on soil fumigated for nematodes prior to propagation.

HELPFUL REFERENCES

Mircetich, S. M., H. W. Fogle, and E. L. Civerolo, 1970. Peach stem pitting: transmission and natural spread. Phytopathology 60:1329-1334.

Smith, S. H., R. F. Stouffer, and D. M. Soulen. 1973. Induction of stem pitting in peaches by mechanical inoculation with tomato ringspot virus. Phytopathology 63:1404-1406.

Necrotic Ring Spot

Before the development of virus indexing programs, necrotic ring spot virus occurred in nearly all orchard and nursery cherry trees. It was also present in peach and plum to a lesser extent. The disease is observed in the orchard primarily on sweet and sour cherry trees. On sweet cherry, the disease is known as "tatter" leaf. In years when symptoms of necrotic ring spot are acute, yields are reduced markedly. Losses for individual trees range from 25 to 50%. Severe strains of the virus may reduce yields over 50%.

Symptoms

Symptoms of necrotic ring spot are usually present on only a few branches of a given tree the first year infection occurs. The next year, other branches show

Fig. 119. Trunk of peach tree with bark removed to show pitting of the wood from stem pitting disease.

Fig. 120. Trunk symptoms of stem pitting on peach. Note thickened bark and wood pitting extending to ground level *(courtesy S.M. Mircetich, USDA).*

symptoms, but the area infected initially may not. In the years symptoms occur, the disease is in the acute or "shock" stage. This is when greatest yield reductions occur. Later, the trees appear normal unless they develop sour cherry yellows.

Individual infected branches may be detected in early spring by delayed foliation and blooming. Leaves on affected branches are small and often have depressed fine lines (etching) and partial-to-complete rings on the upper surface. Leaf symptoms are most pronounced during the 2-wk period following petal fall. Areas of the leaves may become necrotic and fall out, giving the leaves a tattered appearance. Leaves unfolding later often do not show symptoms. Green fruit sometimes show similar arcs and rings.

Terminal and lateral shoots of affected branches are very irregular in length, often stunted or with dieback of the growing points (Fig. 121). Occasionally, large areas of bark are killed and show gumming. Severely affected limbs are also more susceptible to low temperature damage in winter.

Disease Cycle

As a result of nursery certification programs, new trees are virtually free of the disease. Virus movement into young plantings occurs primarily through infected pollen. Thus, trees remain free of ring spot until they begin to bloom heavily. The rate of spread and buildup in a new planting is related to proximity to older, infected trees.

Once transmission occurs, a few weeks to 2 yr are required before symptoms develop. Severity of the symptoms depends upon temperature. Leaf symptoms develop readily between 68 and 75°F, but may occur between 50 and 82°F. Dieback of shoots and spurs is more severe at higher temperatures.

Control

Necrotic ring spot may be reduced through the same procedures described for sour cherry yellows.

HELPFUL REFERENCES

Nyland, G., R. M. Gilmer, and J. D. Moore. 1976. "Prunus" ring spot group. pp. 104-132. *In* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America. U.S. Dept. Agr., Agr. Handbook 437.

Fulton, R. W. 1959. Purification of sour cherry necrotic ringspot and prune dwarf viruses. Virology 9: 522-535.

Fig. 121. Shock symptoms of necrotic ring spot on sour cherry. Note dieback of terminal shoots. Leaves on affected branches often exhibit small, partial-to-complete rings "etched" on the upper surface.

Sour Cherry Yellows

Cherry yellows is currently the most serious disease of Montmorency sour cherry in the Great Lakes fruit belt. It affects trees in such a subtle way that damage is often overlooked until the orchard has become worthless. Initial losses from yellows are not spectacular as with ring spot virus, but increase with time. Yields of sour cherry trees infected while young are reduced by 40 to 50%. The longer a new

Fig. 122. Severe yellowing and defoliation of sour cherry due to sour cherry yellows.

planting can be kept virus-free, the closer it will come to reaching its maximum yield potential.

Symptoms

Cherry yellows derives its name from the characteristic yellowing of the leaves (Fig. 122) followed by heavy leaf drop. Some leaves drop without first turning yellow, but most show different stages of yellow-green mottling. Defoliation generally occurs in waves beginning 3 to 4 wk after petal fall. Severity of each wave depends upon the temperature 30 days prior to the period of defoliation. Low temperatures increase later symptom development.

Yellows-infected trees develop an excess of flower buds on terminals and lateral shoots. In a few years, the bearing surface is reduced since no lateral vegetative buds remain to produce new fruit spurs. The tree exhibits a willowy-type growth with long, bare spaces lacking fruit or spurs.

Flower buds on yellows-infected trees are weaker and abnormally susceptible to low-temperature damage. Bloom is often staggered, leading to uneven fruit ripening. Fruit produced on yellows-infected trees are larger than normal; and because of their size, they may be objectionable for processing. Sweet cherry trees are also infected by yellows, but no symptoms are produced.

Disease Cycle

Transmission of this disease in the orchard is similar to necrotic ring spot, with one major exception. Before a tree exhibits yellows symptoms, it must be infected with necrotic ring spot virus. The disease is caused by a complex of two viruses—possibly more.

Spread of the cherry yellows virus complex follows 2 to 3 yr after the spread of necrotic ring spot. Spread is more rapid where high numbers of infected trees are present.

Control

Sour cherry yellows eventually moves into a disease-free orchard unless removed prematurely for other reasons. Care in planting a new orchard, to reduce the movement of ring spot and yellows into the planting, will pay dividends through increased yields and extended orchard life.

The following program has been shown to retard the movement of ring spot and yellows into cherry plantings. Failure to follow any one practice will defeat the objectives of the program.

Purchase virus-free trees only. Modern nurseries obtain seeds for rootstocks from indexed trees and use virus-free buds to produce trees as free as possible from known viruses. Most states have inspection programs and certify trees indexed free of known viruses. Trees not certified for freedom of viruses should be refused.

Isolate plantings. New orchards should be planted at least 100 ft (preferably 500 ft) from existing cherry blocks. Spread of ring spot and yellows is rapid when older, infected trees are nearby. One way to isolate blocks is to plant apple or pear between cherry blocks.

Plant solid blocks. Replanting missing trees is only profitable for the first 5 yr. Thereafter, avoid replanting. When the orchard becomes unprofitable, remove the block completely.

HELPFUL REFERENCES

Gilmer, R.M., G. Nyland, and J. D. Moore. 1976. Prune dwarf. pp. 179-190. *In* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America. U.S Dept. Agr., Agr. Handbook 437.

Gilmer, R. M. and R. D. Way. 1960. Pollen transmission of necrotic ringspot and prune dwarf virus in sour cherry. Phytopathology 50:624-625.

Green Ring Mottle

Green ring mottle is a virus disease commonly found in older sour cherry orchards established before virus-indexed trees were available. It also occurs as a latent virus in peach, apricot and sweet cherry.

Symptoms and Disease Cycle

Infected sour cherry trees develop yellow leaves similar to sour cherry yellows except for the presence

Fig. 123. Healthy Montmorency cherry leaf on left. Typical symptoms of green ring mottle virus are dark-colored arcs and rings on yellow background. "Constricting Chlorosis" symptom is in second leaf from left *(courtesy K.G. Parker, Cornell Univ.).*

of prominent dark green blotches or rings (Fig. 123). Leaf symptoms often occur in waves extending from late June to mid-July, sometimes later. Defoliation is generally not as severe from green ring mottle as from cherry yellows. Both diseases may be present in the same tree and show symptoms either concurrently or separately. A smaller number of leaves may show yellowing along a lateral vein and exhibit slight leaf distortion at the tip of the vein. This is called "constricting chlorosis" and is often present before the yellow-leaf symptom appears.

Fruit on Montmorency trees with green ring mottle are indented with streaks of dead tissue extending to the pit. The fruit from these trees are often worthless.

Very little is known about how the virus spreads other than through infected propagating materials. In orchard plantings, the disease spreads slowly and is not related to the spread of ring spot and yellows. Weed hosts are known, but their role in spread is not known. The virus is not transmitted by seed.

Fig. 124. Constriction disease of Stanley prune. Note shoots from bud union and smaller size of Myrobalan rootstock compared with the trunk.

Control

Control of green ring mottle virus of stone fruits is based exclusively on starting with nursery stock indexed-free of known viruses and elimination of any tree with disease symptoms.

HELPFUL REFERENCES

Barksdale, T. H. 1959. Green ring mottle virus as an entity distinct from sour cherry ring spot and yellows viruses. Phytopathology 49:777-784.

Parker, K. G., P. R. Fridlund, and R. M. Gilmer. 1976. Green ring mottle. pp. 193-199. *In* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America. U.S. Dept. Agr., Agr. Handbook 437.

Constriction Disease of Stanley Prune

This disease is a problem on Stanley prune trees propagated on certain selections of myrobalan rootstock. Incidence of the disease was reduced but not eliminated when nurseries were provided with selections of myrobalan less prone to the disease than other selections.

Affected trees exhibit smaller-than-normal pale green to yellow leaves and reduced tree growth. They decline for 1 to 3 yr before death occurs. Declining trees often sucker heavily from the myrobalan rootstock. When the soil is removed around the trunk of these unthrifty trees, the shank of the myrobalan rootstock is often restricted on one side and is smaller in diameter than the Stanley prune trunk immediately above the union (Fig. 124). The exact cause of this disease is unknown.

HELPFUL REFERENCES

Brase, K. D. and K. G. Parker. 1955. Decline of Stanley prune trees. Plant Dis. Rep. 39:358-362.

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